ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 49 DECEMBER, 1940 No. 4

LXXI

INTRANASAL VACCINE SPRAY: ITS USE IN PROPHYLAXIS AGAINST THE COMMON COLD*

THEODORE E. WALSH, M.D.

ST. LOUIS

The importance of the common cold in the general health and economy of the community can hardly be exaggerated. Not only are colds, per se, responsible for considerable loss of time and money, but upper respiratory infections initiate many of the more serious diseases. Collins,[1] in a survey of the causes of illness, found that in families in all circumstances in eighteen states colds and bronchitis were twice as frequent as any other disease, both in respect of time lost from work and of confinement to bed. Although in some cases a cold may with prompt care be aborted, the vast majority of colds run a course of from five days to three weeks, and the sufferer is relatively disabled.

It is not surprising, therefore, that considerable attention has been devoted to the prophylaxis of the common cold, and many papers have been written on the use of bacterial vaccines in its prevention. Although there are some who have been favorably impressed with the effect of vaccination the majority of writers have been disappointed in its results. This is the more apparent when

*From the Department of Surgery, Division of Otolaryngology, The University of Chicago.

MEDICAL LIBRARY

large numbers of patients have been treated and the results checked adequately by controls. In 1935, I[2] made a preliminary report on the use of vaccine by intranasal spray. This report concerned the results of the use of intranasal vaccine on a total of 81 patients over a period of two years and was entirely uncontrolled; the results however seemed to warrant the continuance of the study. This paper is a further report of the use of vaccine by intranasal spray with a discussion of the etiology of the cold and a brief review of the literature on the use of vaccines in the prevention of colds.

ETIOLOGY

The nose, lined as it is with ciliated epithelium covered with a protective blanket of mucus, is under favorable circumstances adequately protected against the invasion of pathogenic micro-organisms. It is the breakdown of normal defenses that allows organisms to enter the subepithelial tissues giving rise to the symptoms that we designate as "a cold". The important factors in the defense mechanisms are the continuity of the mucous blanket and the continued activity of the cilia which move it. It is difficult for bacteria to find their way through an intact mucous covering. The breaking of the mucous coat and the exposure of the underlying epithelium opens a portal of entry for pathogenic micro-organisms.

The factors that are responsible for the breaking of the layer of mucus and for the cessation of ciliary activity are numerous and may be described as (a) biologic, (b) metabolic, and (c) physical.

Biologic.—There can be no doubt that a filter-passing virus is responsible for the initiation of the common cold in a certain percentage of cases. Kruse,[3] Foster,[4] Dochez and his co-workers[5] and Long and his associates[6] have all transmitted colds to normal individuals by the intranasal inoculation of filtered washings from the nasopharynx of others with acute colds. These workers were successful in their transmissions in about 40 to 60 per cent of cases. The patients under observation were kept in strict isolation with no chance of secondary infection from the outside world. There was no significant change in the flora of the nose during the course of the colds initiated by the virus. The colds were of short duration, uncomplicated and self-limiting. The chief clinical symptoms were stuffiness of the nose, coryza and sneezing. In several cases it was noted that there was an exacerbation of the cold when the individual was freed from quarantine after apparent recovery from the experimental cold. Bloomfield[7] investigated the flora of the nose and throat in normal individuals and in those with cold and states: "In

the uncomplicated cases the outstanding fact was this, that the flora differed in no fundamental way from that which obtains in healthy individuals. As in normal controls, non-hemolytic Streptococci and Gram negative cocci are constantly present. As in the normal there is a shifting transient flora consisting of various organisms—diphtheroids, coarse Gram positive cocci, minute Gram positive organisms, white Staphylococci and others. Two points in regard to this transient flora are, however, worthy of note. It is distinctly richer and more varied than that found in our series of controls. . . . A conceivable explanation of these findings is that the disturbance of the mucous membranes during the cold allows a general increase in the activity of bacterial growth on these surfaces. This fits in with the old theory that the cold, whether produced primarily by cold or by infection, leads to environmental changes which, as it were, light up the bacterial flora already present in the mouth." He concludes that "The primary cause of colds is probably an organism as yet unknown and certainly not one of the usual pathogens such as Streptococcus, Pneumococcus, B. influenzae or Staphylococcus. But the primary cold, whatever its final cause, alters the mucous membranes in such a way as to allow secondary bacterial invasion and consequent frequent development of complications." Shibley, Hanger and Dochez[8] made a similar study and found that certain organisms were prominent in colds, usually as late or secondary invaders; these included Staphylococcus aureus, Streptococcus haemolyticus and B. influenzae. Dochez,[9] speaking of the mechanism of upper respiratory infections says: "the conclusion may be drawn that one type of acute upper respiratory infection is caused by a filterable virus. In addition to initiating the symptoms of infection, this agent may be considered to provoke increased activity of any potential pathogens that may happen to be present in the respiratory tract, and furthermore to render the respiratory tract susceptible to implantation of pathogenic organisms from the exterior."

It seems reasonable therefore to suggest that the virus alters the normal defenses of the nasal mucous membrane and permits secondary bacterial invasion. The cold that results is mild or severe depending on the relative pathogenicity of the secondary invaders and the susceptibility of the individual attacked. It is the secondary invaders that are responsible for the complications and sequelae that are so disabling. Whether the virus can penetrate the normal defenses of the nose if the nose is subjected to ideal conditions is open to question. The experiments of Kerr and Lagen[10] tend to show that this may be so. They kept 19 subjects who were susceptible to colds as shown by

their previous histories, for a period of observation in an air conditioned room in which the temperature was maintained at 70 degrees F. and the humidity at 55 per cent of saturation. Into this room, after a period of observation, were admitted persons with acute colds. Exposure of the subjects to the colds was intimate and was prolonged for several hours. Drinking glasses and mouth thermometers were contaminated with fresh secretions from the individuals with colds, and in some instances fresh secretions from the colds were injected into the conjunctival sacs of the experimental subjects. Nineteen individuals were so treated in five groups and so exposed to five different colds, and in no case was a cold contracted by the exposed individuals. The authors conclude: "This does not prove that there is no type of rhinitis which is contagious, but it does suggest that colds are not so readily transmitted as many assert." I suggest however that the conclusion to be drawn from this experiment is that under favorable conditions the nasal defenses are adequate even against the virus.

Metabolic.—There can be little doubt that factors which alter the normal metabolic processes of the body affect the membranes of the upper respiratory tract, either by alteration in the activity of the cilia and the production of mucus or in the immunologic response of the membrane. Fatigue, chilling, exposure, overindulgence in food or drink, dietary insufficiencies and glandular disturbances all have their effects. Probably all of us have experienced the nasal congestion that follows a too heavy meal or the assimilation of alcoholic drinks. Rhinologists are familiar with the alteration in the nasal mucosa in thyroid diseases and in women at the menstrual period. These factors impair the efficiency of the nasal defenses and permit infection of the subepithelial tissues by pathogens that may reach the nose at such times.

Physical.—The work of Proetz and Hilding has thrown much light on the effects of temperature and of humidity on the nasal mucous membrane. Proetz[11] showed that cilia beat effectively at between 18 and 33 degrees C. He observed the cilia both in living rabbits' sinuses and also in extirpated human membranes and writes: "At 35 degrees C. it became necessary to supply fluids with increasing frequency owing to evaporation. At 40 degrees C. motion was greatly retarded and at 43 or 44 degrees C. it ceased entirely." He also states that the respiration of cold air does not damage cilia, especially in view of the fact that inspired cold blasts are alternated with expired air at 37 degrees C. Anyone who has observed ciliary activity either in the living animal or in extirpated human membranes

cannot help but be impressed with the effect of drying on the activity of the cilia.[12] Cilia slow immediately, and cease to beat in a very short time if they are not kept moist and recover their activity only after a considerable period of moistening.

Hilding[13] has studied the physiology of drainage of nasal mucus and describes active and inactive areas in the nose. The epithelium of the inactive areas is non-ciliated. The mucus which covers the inactive areas of the nose is moved by the traction of the mucus on the active areas. Although the tensile strength of nasal mucus is considerable, a firmly adherent crust of mucus will lead to a break in the mucus blanket posterior to it, and this exposes the epithelium to any bacteria that may be in the nose. Hilding also described the changes that occur in the morphology of the epithelium when it is exposed to increased air flow over it. He found that in dogs in which one nostril had been occluded the epithelium of the open nostril lost its cilia and became squamous in type. The same change can occur where, owing to septal or other anatomic deformities, the normal airflow through the nostril is altered and a jet of air impinges constantly on one portion of the mucosa. The resultant drying and the loss of ciliary activity break down the normal defenses of the nose. The same effect may result from swelling of the turbinates due to vasodilatation following alcoholic excesses and so on.

To summarize, therefore, any one of many factors—biologic, metabolic or physical—may be instrumental in breaking down the normal defenses of the nose and predispose to the production of a cold. Whether this develops or not depends on the presence of pathogenic organisms and the resistance of the individual to them.

The importance of the metabolic and physical factors in initiating colds is illustrated by the seasonal peaks of colds that are reported by so many observers. These peaks occur in October and November, in the early part of January and again in March or April. In Chicago I have noticed that the first epidemic of colds is always associated with the first cold days in the fall. Heat is turned on in the buildings, and the humidity in the rooms drops to between 13 and 20 per cent of saturation. The nasal membranes become dry, and the defenses are broken down. The second peak of colds seems to be associated with the holiday season. At this time normal routines of life are disrupted. Excesses of eating and drinking are associated with the festivities of Christmas and the New Year, and much sleep is lost by the average individual at this time. Colds follow. In the March-April period, spells of sunny warm weather are not uncommonly followed by a sudden return to cold damp days. The

unwary is apt to doff his overcoat and warm clothes and becomes chilled. A cold often follows. There is possibly much reason for the old English proverb which says: "Cast ne'er a clout till May be out, buckle to the chin till May come in."

PROPHYLAXIS

The ways of modern life, particularly in large cities, with conditions of overcrowding, imperfect heating of buildings and the fatigue and nervous tension associated with the daily round of affairs make it impossible to maintain an adequate first line of defense against common respiratory infections. There remains then the necessity of building an adequate second line of defense so that in the event of organisms gaining access to the subepithelial tissues they may be held locally and destroyed before they have a chance to grow and spread to adjoining tissues. It is to this end that vaccines of the common respiratory pathogens have been advocated for years. Although there have been reports of benefit from vaccination in some of the many series reported in the literature, one is struck by the fact that in all series that were adequately controlled the authors found little benefit from the use of vaccine as a prophylactic against the common cold. The vaccines have consisted of a mixture of the common upper respiratory pathogens and have been administered either subcutaneously or, more recently, by mouth.

D. and R. Thomson[15] survey the literature prior to 1932. They say: "In our opinion there is abundance of irrefutable evidence in favor of the value of vaccines, both toxic and detoxicated, in the treatment of colds and catarrhs of the respiratory tract. Preventive inoculations against colds give no guarantee of immunity to catarrhs, but it is our experience that colds contracted in inoculated persons are less severe as a rule than those contracted by the uninoculated, and furthermore the inoculated are less liable to complications." Von Sholly and Park[16] however, who gave injections of vaccine to 1536 persons and had as controls 3025 uninoculated persons, state: "On the whole our evidence does not make a strong case in favor of the vaccine given by us as a prophylactic agent against acute respiratory diseases—pneumonia alone excepted." Jordan and Sharpe[17] had some 6000 persons under observation in 1921. One half were vaccinated; the rest were untreated. They conclude: "Rhinitis and bronchitis developed with about equal frequency in the vaccinated and unvaccinated groups." Blitch and Doyle[18] used a stock vaccine for the prevention of colds in soldiers at Fort Monmouth, New Jersey, in 1936-37. They vaccinated 375 men and had 475 as controls. The

total admission to sick report (rate per 1000 men) was 209 for the vaccinated and 224 for the unvaccinated, and the average days lost from duty per man was 5.9 for the vaccinated and 6.1 for the unvaccinated. They conclude that vaccination does not prevent colds or their complications nor does it cut down the number of days lost on account of colds. Diehl, Baker and Cowan[19] give an excellent report on a carefully controlled series of vaccinations during the years 1936-37 and 1937-38. Their subjects were students at the University of Minnesota who volunteered to participate in the study because they were particularly susceptible to colds. The vaccines were given subcutaneously to some and by mouth to others. The controls were treated in the former instance with injections of sterile physiologic saline at the same intervals as the vaccine was administered and in the latter by capsules containing lactose. Their tables are extremely interesting. They show a marked reduction in the number of colds during the experimental years over those reported in the year prior to the experiment, not only in the vaccinated groups but also in the controls. The reduction in the number of colds was approximately the same both in the vaccinated and in the control groups, independent of the nature of the vaccine used. The authors remark: "One of the significant aspects of this study is the great reduction in the number of colds which the members of the control groups reported during the experimental period as compared to the number the same students reported for the previous year. In fact, these results were as good as many of those reported in uncontrolled studies which recommend the use of cold vaccines." Hauser and Hauser[21] in a study with students at the University of Michigan confirm the findings of Diehl and his co-workers. Sherman[20] in 1938 tried several methods of cold prevention among the employees of Cadbury's factory at Bourneville, England. The treatments used were Entoral (238 subjects), capsule of Vitamins A and D (330 subjects), subcutaneous vaccine (213 subjects) and exposures to ultra-violet light (236 subjects). There were 593 controls observed. The only procedure that seemed to have any benefit was the ultra-violet light, and the author says: "There seemed to be an increased susceptibility in patients on Entoral and subcutaneous vaccines."

The results of vaccination by subcutaneous and oral methods against colds are disappointing. The question arises: Wherein lies the cause of the failure? Is it that the upper respiratory pathogens are poor antigens, or is it perhaps that in spite of an increase of circulating antibodies against the pathogens the portal of entry of these organisms is still left unguarded? It is generally agreed that the ideal

conditions for tissue immunity are those in which there are not only sufficient specific antibodies but also an accumulation of phagocytes in the tissue to be guarded. Cannon and I[22] showed that such a condition can be accomplished in the nose by the simple application of a vaccine to the nasal mucosa. We found that following the local application of a vaccine of Bact. paratyphosum B. in the nose of rabbits, the agglutinin titer of the nasal tissues was frequently as high as that of the blood serum and constantly higher than that of other tissues such as spleen, liver and gut; also that the nasal tissues of animals so vaccinated showed a subepithelial accumulation of macrophages and a hyperplasia of lymphoid tissue. These changes were not present when the vaccine was administered by subcutaneous injection or by mouth. We also showed[23] that in animals immunized against two different antigens simultaneously, the one by intranasal application, the other by parenteral injection, the ratio of tissue antibodies to serum antibodies (T/S ratio) was constantly higher for the locally applied antigen than for that administered generally. On the average the T/S ratio for parenterally administered antigens is from 1:10 to 1:15 whereas that for locally applied antigens is 1:5. This accumulation of specific antibodies in an area in which there is an accumulation of phagocytic cells enhances the resistance of tissue and should constitute an adequate barrier against the invasion of pathogenic micro-organisms. That such an increased resistance to infection does obtain is shown by our experiments[24] and those of Bull and McKee[25] and Linton[26] in which, following nasal vaccination, there occurred an increased resistance to intranasal infection by a virulent organism, this in spite of the fact that no specific antibodies could be demonstrated in the blood stream.[24, 25]

It seems reasonable to assume from these experiments that if any protection against colds from the use of vaccines is to be hoped for, the rational method of immunization is by application of vaccine to the nasal mucosa. Because of the specificity of the immune reaction and because of the variety of possible upper respiratory pathogens it seems essential that the vaccine should be polyvalent and that fresh strains of organisms should be added to the stock from year to year.

METHODS

The vaccines were prepared in the following manner. Cultures were obtained in the Ear, Nose and Throat Clinic from various patients with acute and chronic infections of the upper respiratory tract and ears. Cultures were planted on blood agar plates and incubated at 37 degrees C. for from 24 to 48 hours. Typical colonies were

picked from such plates for identification and for pure culture, planted in mediums suitable for quantity growth and incubated at 37 degrees C. for from 24 to 72 hours. The mediums used were blood agar and dextrose veal infusion broth. The organisms were removed from the agar plates by scraping and from the broth by centrifugation and were killed by suspension in a physiologic solution of sodium chloride with formaldehyde U. S. P. 0.4 per cent and incubation at 37 degrees C. for four days. This suspension was centrifugated and the organisms washed two or three times in physiologic salt solution to remove all traces of formaldehyde. The organisms were then suspended in physiologic salt solution in a volume approximately ten times that of the packed bacterial bodies. These suspensions represented a concentrated stock vaccine of the various organisms obtained. The organisms thus obtained were Staphylococcus albus hemolytic and non-hemolytic, Staphylococcus aureus hemolytic, Streptococcus alpha, beta and gamma, Pneumococcus Types I, II, III and Group IV, diphtheroid bacilli, Friedlander's bacillus and Bacillus influenzae. Fresh cultures were added to the stock from time to time as cases presented themselves in the clinic. The concentrated vaccines were then mixed in the following proportions:

	Parts
Staph. albus—hemolytic and non-hemolytic, of each	1
Staph. aureus—hemolytic	1
Strep. a, b, and g, of each	2
Pneumococci, all types, of each	1
Diphtheroid bacilli	1
Friedlander's bacillus	2
B. influenzae	2

The mixture was diluted with physiologic salt solution so that the turbidity of the final suspension was approximately equal to that of one billion Staphylococci per cubic centimeter of suspension. Merthiolate 1:10,000 was added as a preservative. The final suspension was tested for sterility both aerobically and anaerobically and was dispensed in rubber stoppered bottles.

For the use of the vaccine the patients were advised at first to purchase a De Vilbiss No. 20 nasal atomizer. This is an inexpensive atomizer but has the disadvantage that as the nasal tip is continuous with the bottle, secretions from the nose are apt to flow back into the atomizer and contaminate the vaccine. Latterly a De Vilbiss No. 28 has been used. This is conveniently small, is easily kept clean and owing to its construction is more suitable than the No. 20. The patients were instructed how to sterilize the atomizer before filling

it with vaccine and were advised to put only a small quantity of vaccine in the atomizer at a time and to keep the remainder well corked in the refrigerator. Six puffs of the vaccine were to be snuffed up into each nostril every night just before retiring. This was to be done each night for three weeks and again after one week's rest for two weeks and continued for alternate two weeks' periods for the remainder of the "cold year" (September to May).

It was emphasized to the patients that the vaccine is a suspension of bacterial bodies in saline and that to be effective the bacterial bodies must reach the nose. They were instructed therefore that the bottle must be shaken well before the atomizer is filled and that the atomizer should be shaken before the nose is sprayed. The necessity for this emphasis was impressed on me when one patient, an eminent biologist who had been using the vaccine, developed a severe cold, and on questioning him I found that he had never shaken the vaccine, neither before filling his atomizer nor before using it. He had, in fact, been spraying his nose carefully with simple saline solution! There was no wonder that the therapy failed in his case.

The patients studied were those who presented themselves at the clinic for diseases of the nose and throat, complaining of frequent colds in winter. Every effort was made to determine that the "colds" were true infections and not vasomotor reactions so often called "colds" by the laity. The patients were carefully examined to rule out chronic sinus infections, infections of the tonsils and other conditions as etiologic factors of the colds. Only those who showed no chronic nasal or faucial pathologic changes but who were subject to frequent infections of the upper respiratory tract were studied for this report, with certain exceptions that are mentioned later. Where possible, the patients were seen at intervals during the "cold year" (September to May) or at the end of it. Where this was not possible a form letter was sent to patients asking them the following questions:

1. Have you had any colds since using the vaccine? If so, how many?
2. Are they any different from the colds you had previously?
   (a) More severe?
   (b) Less severe?
3. Have there been any complications of the colds, such as bronchitis?
4. Do you believe that the vaccine has helped you or not?

RESULTS

In recording the results of the use of a vaccine in the prevention of colds it has been the custom of most writers to record the total number of colds in a group of individuals in the year before the vaccine was used and again in the year in which the vaccine was given and thereby arrive at the average number of colds per person before and after vaccination. The percentage reduction in the number of colds in such a group of individuals has been taken as the criterion of the efficacy of the vaccine. I have felt that this is an inaccurate method of recording results. I have found that on the whole patients are very vague as to the exact number of colds they have experienced in previous years. Replies to the question "How many colds have you had in previous years?" have been "three or four" or "six or eight" or "one after another" or "many" or sometimes "continuous". Diehl remarked how unreliable were the memories of his students in this regard and pointed out how misleading the percentage reduction in colds might be except in a carefully controlled series of cases. For this reason I have recorded my results as "good", "fair" or "bad" with the following criteria. It must be reiterated that all patients who were given vaccine were susceptible to colds and were said to have had three or more severe colds a year in previous years. If then they had no colds or not more than two mild attacks of rhinitis lasting not more than 48 hours the result was said to be "good". More than two attacks of rhinitis even though of short duration and not more than three such attacks, or one or two mild colds, constituted a "fair" result. More than two mild colds or even one severe cold, in spite of the patient's protestations of improvement over previous years was labelled a "bad" result. The results are seen in Tables I and II.

TABLE I

Vaccine Used For	By Patients	Good	Fair	Bad
1 year only	274	137	34	76
2 years only	76	61	8	7
3 years only	40	38	1	1
4 years only	11	10	1	0
5 years only	6	5	1	0
6 years only	2	2	0	0
7 years only	2	2	0	0
Total	384	255	45	84

TABLE II

Year	No. of Patients	Good	Fair	Combined	Bad
1932-33	24	19—79%	1— 4%	20—83%	4—17%
1933-34	66	45—68%	8—12%	53—80%	13—20%
1934-35	79	63—80%	8—10%	71—90%	8—10%
1935-36	100	76—76%	12—12%	88—88%	12—12%
1936-37	108	80—74%	13—12%	93—86%	15—14%
1937-38	101	78—77%	7— 7%	85—84%	16—16%
1938-39	90	65—72%	10—11%	75—83%	15—17%
1939-40	59	38—66%	6—10%	44—76%	15—24%
Totals	627	464—74%	65—10%	529—84%	98—16%

It is seen from Table I that the total number of patients who were treated with the vaccine in the eight years from 1932 to 1940 inclusive was 384. Of these, 247 used the vaccine for one year only; 137 (56%) had a good result, 34 (14%) a fair result and 76 (30%) were not helped. However, of the 137 patients who used the vaccine for two to seven years only 8 (6%) were not benefited.

Table II shows the results obtained in the various years. Of a total of 627 patient years of vaccine there were 74 per cent good results, 10 per cent fair results and 16 per cent failures. It has been our experience that it is difficult to get replies to questionnaires promptly from patients as to their results from vaccination. The questionnaires are sent out in May and a reply requested, but in many cases no word has been obtained from the patients until the following September when they returned for more vaccine. On the contrary if the patient was not benefited from the vaccine he seldom hesitated to write and say so. This perhaps accounts for the poorer results in 1939-40 than in other years, as at the time of writing only 59 of 101 replies have been obtained. Another possible reason for the greater percentage of failures in the last year is that there was a severe epidemic of influenza in Chicago. The vaccine has no protective value against virus infections, and it is difficult for patients to differentiate between a common cold and a virus infection.

During the year 1939-40 I supplied the vaccine to physicians both in Chicago and in towns in Illinois and Wisconsin and asked them to let me know what results they obtained with the vaccine in

their practices. Twelve physicians co-operated in this with me and used the vaccine on a total of 69 patients. Of these 69 patients 45 (65%) had good results, 12 (17%) had fair results, and 12 (17%) were not benefited. These results are those recorded by the physicians.

During the season 1934-35 I was enabled, through the courtesy and co-operation of Dr. Dudley B. Reed and his staff in the Student Health Service of the University of Chicago, to make a controlled experiment. Dr. Reed let it be known that his service could offer prophylaxis against colds to any who were susceptible. I supplied Dr. Reed with two solutions labelled "A" and "B". "A" was the vaccine and "B" a suspension of starch in saline with 1:10,000 merthiolate added. Grossly, by sight, taste or smell, it was impossible to differentiate between the two solutions. The physicians who dispensed the solutions did not know which was the vaccine and which the control. Students who applied for prophylaxis were given alternately "A" and "B". They were required to report at the student health clinic monthly during the season and at the end of the season were sent a questionnaire similar to that used in the Otolaryngology Clinic. I did not see any of the subjects and only had the reports and letters from which to evaluate results. One hundred forty-eight students were thus observed, 74 who used vaccine and 74 the control. Table III shows the results.

TABLE III

	No. of Patients	Results			
		Good	Fair	Combined	Bad
Vaccine	74	46—62%	11—15%	57—77%	17—23%
Control	74	37—50%		37—50%	37—50%

This experiment was designed to eliminate any possible bias on my part in the evaluation of results. I saw none of the subjects of the experiment. The physicians in the Student Health Service, even if they had been inclined to be enthusiastic about intranasal vaccination, were in ignorance as to which was the vaccine and which the control, and the students were not aware that an experiment was in progress. It is interesting to note 50 per cent good results in the control group. It bears out Diehl's observations. In the control group however there were no "Fair" results. Either the patients had no colds or they had just as many and as severe colds as before. The 15 per cent "fair" results in the vaccinated group is, I believe, signifi-

cant. These results, although not quite so good, approximate the results obtained in the clinic patients.

The following case reports are typical of many others and tend to constitute a further "control" in this study.

REPORT OF CASES

CASE 1.—O. H. R., male, aged 49, a physician, gives the following history of upper respiratory infections:

1934-35—Several colds during the winter and one followed by prolonged respiratory infection necessitating two weeks in hospital.

1935-36—Same as previous year.

September 1936—Sore throat followed by laryngitis lasting 10 days.

Vaccine begun October 1, 1936.

1936-37—No colds or other respiratory infection.

1937-38—Vaccine and no colds or respiratory infection.

Stopped vaccine May 1, 1938. September, 1938, had a cold, not very severe. Otober, 1938, another cold. Began vaccine latter part of October, 1938. No further colds during the winter of 1938-39. Again discontinued vaccine May 1, 1939. September, 1939, had a cold, after which he began vaccine therapy. No colds to date (May 1, 1940).

CASE 2.—H. R. W., male, aged 40, teacher. Gave a history of having four or five bad colds a year. Started vaccine October 28, 1935. Had one very mild cold during the winter 1935-36. Did not use vaccine during 1936-37 and had four bad colds. Started vaccine again on September 15, 1937, and had two very slight colds during 1937-38 and again the same result during 1938-39 with vaccine. Used vaccine again during 1939-40 and had no colds.

CASE 3.—J. C., male, aged 21. Came to the clinic complaining of many severe colds each winter. Was started on vaccine October 4, 1933, and had no colds during that winter. He did not use vaccine in the winter of 1934-35, and starting in November, 1934, he had many colds until December, 1935, when he returned to the clinic requesting more vaccine. Started vaccine again December 2, 1935, and used it during 1936, 1937 and 1938, and was free from colds. He stopped using vaccine again in March, 1938, and six weeks later developed a cold.

CASE 4.—G. R., female, aged 32. Was first seen in 1932 with a chronic bilateral maxillary sinusitis. Antrum windows were made and the sinusitis cleared up. Following this however the patient had frequent severe acute colds and in November, 1934, was given a vaccine. She had no colds during the winter of 1934-35. She used no vaccine during the 1935-36 season and again had two severe colds. In 1936-37 she again used vaccine and again had no colds. During 1937-38 and 1938-39 she did not use vaccine and each year had a severe cold in March.

CASE 5.—R. D., male, aged 12, was brought into the clinic because of frequent colds and resulting loss of time from school. He had been treated in previ-

ous years by subcutaneous vaccination with no benefit. He used the nasal vaccine during the 1933-34 season and had two mild colds lasting a day each with no loss of time from school.

CASE 6.—J. M., male, 22, student. Complained of almost continuous colds in winter. In 1937-38 he was given the intranasal vaccine and had no colds. In the winter of 1938-39 he was given injections of "Immunogen" in the Student Health Clinic and had frequent bad colds.

CASE 7.—J. H. M., male, 22, student. Was in the experimental group during 1934-35. He gave a history of having three or four bad colds during a winter. During 1934-35 he was in the control group and had four severe colds. In 1935-36 he was sent to the Clinic from the Student Health Service for vaccine and used it during that year and had no colds.

CASE 8.—B. L., male, aged 18, student, was in the experimental control group of the Student Health Service during 1934-35 and had four severe colds. In 1935-36 he was given the vaccine in the E. N. T. Clinic and had no colds.

CASE 9.—M. B., female, aged 49. Was seen in the E. N. T. Clinic April 16, 1936, complaining of attacks of acute bronchitis three or four times each winter. The attacks were always related to a dryness in the nasopharynx and lasted about three weeks. Examination of the nose and throat was essentially negative, and x-rays of the sinuses and chest showed no pathologic changes. She was given intranasal vaccine and started using it in August 1936. She had no bronchitis that year and has continued the vaccine each year subsequently with complete freedom from bronchitis.

CASE 10.—J. K., male, aged 13. Was seen in the E. N. T. Clinic first in July 1931. He complained of constant colds and had a chronic otitis media, right side. The nose was full of mucopurulent discharge. Enlarged tonsils and adenoids were removed, and because of clouding on x-ray examination a window was made into the right antrum. Neither his ear nor his nose improved, and he was given a series of vaccine injections during 1931 and 1932 with no improvement. I saw him first in September 1932 and started him on an autogenous intranasal vaccine. One month later his nose was dry, and his ear no longer was running. He was continued on intranasal vaccine (stock) and was seen at intervals of two weeks during the remainder of 1932 and until October 9, 1935, and during that time had two colds, one that lasted a week and was mild, the other that lasted two days.

DISCUSSION

It will be generally agreed that the symptom-complex known as "a cold"—nasal obstruction, sneezing, rhinorrhea followed by a period of mucopurulent nasal discharge which lasts from a few days to three weeks—is caused by infection of the upper respiratory tissues by pathogenic bacteria. Such infection occurs only when the normal defenses of the nose and throat break down. It has been suggested above that factors physical, metabolic and biologic are responsible for the failure of the upper respiratory defense mechanisms. The habits and customs of modern civilized life make the control of the physical

and metabolic factors obviously impossible, and as yet there is no known method that is adequate to protect against the virus of the common cold. There remains but one hope in prophylaxis, namely, to increase individual immunity against the pathogenic bacterial invaders.

There is ample experimental evidence to indicate why the parenteral administration of vaccine has produced such disappointing results in protecting against so "local" a disease. The lack of concentration of specific antibodies in the upper respiratory tissues and the absence of mobilized phagocytes at the point of entrance of the invading organisms leave the tissues of the nose and throat relatively unguarded in spite of a possibly adequate quantity of circulating antibodies. If tissue immunity depends on the presence not only of specific antibodies but also of phagocytes, as the experiments of Clark[27] indicate, then the administration of vaccine by injection or by mouth should be expected to fail. Experiments have shown, however that the application of vaccine to the nasal mucosa results in an accumulation in the mucosa both of phagocytes and of antibodies. The results reported here of the use of vaccine locally as a prophylaxis against colds seem to give clinical support to the experimental evidence and to refute Ward's[28] pessimistic statement that "Nothing will protect a susceptible subject from the three principal colds of the year".

It is apparent, however, that the immunity conferred by vaccination against the respiratory pathogens is short lived. Perhaps this is because of the poor antigenicity of the organisms or possibly because antibodies diffuse relatively rapidly out of tissues. We[29] found that in animals vaccinated intranasally against B. paratyphosum B. the agglutinin titer of the nasal mucosa would change from 1:1960 to 1:120 or less in three to six months. It was not uncommon in patients in this series to have a cold develop six weeks to two months after vaccination had been discontinued. It is advisable therefore to start vaccination in late August or early September, well ahead of the time that the first epidemic of colds is expected, and to continue with the vaccination at least until May.

The specificity of the immune reaction and the variety of organisms that can be considered as respiratory pathogens makes the desirability of a polyvalent vaccine obvious. In some of the earlier cases autogenous vaccines were tried. It was found that when infections occurred after vaccination the predominating organisms were other than those in the vaccine used. For the same reason it has

seemed advisable to prepare a vaccine in the locale in which it is to be used and to replenish the stock of organisms from time to time. It seems not unlikely that variations in strains of the same organism could give rise to different immune responses.

In their experimental transmission of colds by means of the filterable virus Dochez and his co-workers[5a] describe the onset of symptoms of sneezing, coryza, nasal obstruction and discharge about 48 hours after inoculation of the virus. The symptoms lasted about four days and were not accompanied by much change in the bacterial flora. These patients were in isolation in hospital. Two of the three patients had a definite exacerbation of their colds two days after leaving the hospital. Many patients in the vaccinated series reported here have had similar experiences when exposed to others with acute colds. They tell of the sudden onset of nasal obstruction, sneezing and rhinorrhea which led them to believe that they were "in for a real bad cold". The symptoms, however, subsided in 48 hours and were not followed by purulent nasal discharge or any of the common complications of a cold. I had such an experience in 1935. My colds will normally run a course of some three weeks, during which time I have profuse mucopurulent nasal discharge and an annoying loss of the senses of taste and smell. Smoking a pipe at this time is virtually impossible because of the alteration of the senses of taste and small. In Christmas week of the year mentioned I was called to see a patient who had an acute cold of about two days' duration. While examining her throat she inadvertently coughed in my face. I had used vaccine since September and felt satisfied that I was probably immune to any cold and so thought nothing of the accident. Next morning, however, I had nasal obstruction and a profuse rhinorrhea and lacrimation accompanied by much sneezing. I felt sure the vaccine had been a failure in my case and that I was in for three weeks or so of nasal discomfort. By the following morning, however, the symptoms had subsided and except for a slightly dry nose I was well. Moreover, except for the one day I was able to smoke a pipe with unimpaired enjoyment. Similar experiences have been described by many of the patients using vaccine. The explanation suggested is that the acute symptoms can be ascribed to the virus. In an unprotected nose a cold of one to three weeks' duration would probably have resulted, but in a nose in which there was enhanced local immunity, secondary infection with respiratory pathogens did not occur. It would be of great interest to see what effect, if any, preliminary vaccination by

means of a spray would have on the experimental transmission of colds in apes and humans as reported by Dochez.

This study is concerned with the effect of intranasal vaccine on the incidence of colds. Except in a few cases the method was not used as a treatment for chronic conditions such as sinusitis and "catarrh", and no adequate evidence is available from which one can draw positive conclusions. Case 10 leads one to think that the method might be of value in selected cases. Where complications such as bronchitis, sinusitis and otitis media commonly follow acute colds and become the patient's chief complaint, this method of vaccination has proved of benefit—merely by preventing the initial infection. Case 9 illustrates this.

In all the cases observed there were only three patients who experienced untoward reactions to the use of the intranasal spray. One patient complained that she became nauseated by the vaccine dripping into the throat when she used it. One patient, a man, had nasal vestibulitis with furuncles each time he used the atomizer. After several attempts to use the vaccine even after sterilizing the atomizer tip he eventually had to give it up. One patient, a woman, who was known to be allergic to the fall pollens and who sought relief for her frequent winter colds, used the vaccine for some months with complete relief from colds, and then suddenly developed hives. The hives were definitely related to the use of the vaccine, and she had to discontinue her treatments. Apart from these there were no reactions either local or general to the use of the vaccine spray.

A method of immunization that depends for its success on the memory and routine habits of the patient is perhaps open to criticism. The results indicate, however, that such criticism is not valid. It is not difficult to establish a routine in which the use of the vaccine becomes a part of the evening toilet. It is suggested that the atomizer be kept in the bathroom and that spraying be the last act before retiring. Whether the spray is used each day in alternate weeks or whether there is no rest period taken is, I believe, immaterial. The important point is that the rest period should be no longer than two weeks.

SUMMARY AND CONCLUSIONS

The etiology of the common cold is discussed and factors biologic, metabolic and physical are considered as primarily responsible for the breakdown of the normal defenses of the nose. It is when the normal defenses of the nose have been overcome that secondary

invasion of the nasal tissues by the common respiratory pathogens occurs and gives rise to the symptoms that are called "a cold".

The prophylaxis of colds by the use of subcutaneous injection of vaccines has proved disappointing. The reasons for the failure of this method of vaccination are discussed. Experimental evidence is offered to show that the rational method of vaccination against so "local" a disease as a cold is by the local application of vaccine to the nasal mucosa.

The results of such vaccination in 384 patients over a period of eight years are given. Of a total of 627 patient-years of vaccination there were 74 per cent good results, 10 per cent fair results and 16 per cent failures. A controlled experiment with 144 patients is described and a few case histories, which also constitutes a control, are given.

It is suggested that the results warrant the conclusion that the method of local vaccination against the common cold offers the best means of prophylaxis at our disposal.

WASHINGTON UNIVERSITY SCHOOL OF MEDICINE.

BIBLIOGRAPHY

1. Collins, S. D.: U. S. Public Health Reports, 28:283 (March), 1933.

2. Walsh, T. E.: Intranasal Vaccine Spray. Arch. Otol., 21:147, 1935.

3. Kruse, W.: Munch. Med. Woch., 61:1457, 1914.

4. Foster, G. B.: Etiology of the Common Cold. J. Infect. Dis., 21:451, 1917.

5. Dochez, A. R.: Shibley, G. S., Mills, K. C.: a) Studies in the Common Cold. J. Exp. Med., 52:701, 1940. b) Studies on the Etiology of the Common Cold. J. A. M. A., 95:1553, 1930.

6. Long, P. H., Doull, J. A., Bowen, J. M., McComb, E.: Etiology of Acute Upper Respiratory Infections (Common Cold). J. Exp. Med., 53:447, 1931.

7. Bloomfield, A.: Variations in the Bacterial Flora of the Upper Air Passages During the Course of Common Colds. Bull. Johns Hopkins Hosp., 32:121, 1921.

8. Shibley, G. S., Hangar, F. M., Dochez, A. R.: Studies in the Common Cold. J. Exp. Med., 43:415, 1926.

9. Dochez, A. R., Mills, K. C., Kneeland, Y.: Studies on the Mechanism of Upper Respiratory Infections. Tr. Assoc. Am. Phys., 46:200, 1931.

10. Kerr, W. J., Lagen, J. B.: Studies on the Transmissibility of the Common Cold. Tr. Assoc. Am. Phys., 49:245, 1934.

11. Proetz, A. W.: Effect of Temperature on Nasal Cilia. Arch. Otol., 19:607, 1934.

12. Proetz, A. W.: Nasal Ciliated Epithelium with Special Reference to Infection and Treatment. J. Laryng. and Otol., 49:557, 1934.

13. Hilding, A.: Physiology of Drainage of Nasal Mucus. Arch. Otol., 15:92, 1932.

14. Hilding, A.: Experimental Surgery of the Nose and Sinuses. Arch. Otol., 16:169, 1932.

15. Thomson, D., and Thomson, R.: The Common Cold. Ann. Pickett-Thomson Res. Lab., Vol 8 (Dec.), 1932.

16. Von Sholly, A. K., and Park, W. H.: Report on the Prophylactic Vaccination of 1536 Persons Against Acute Respiratory Diseases—1919-1920. J. Immunol., 6:103, 1921.

17. Jordon, E. O., and Sharp, W. B.: Influenza Studies. Effect of Vaccination against Influenza and Some Other Respiratory Infections. J. Infect. Dis., 28:357, 1921.

18. Blitch, C. G., and Doyle, J. F.: The Efficacy of the Cold Vaccine. Military Surgeon, 84:46, 1937.

19. Diehl, H. S., Baker, A. B., Cowan, D. W.: Cold Vaccine: An Evaluation Based on a Controlled Study. J. A. M. A., 111:1168, 1938.

20. Sherman, J. B.: Prophylaxis of the Common Cold. Brit. Med. J., 2:903, 1938.

21. Hauser, I. J., and Hauser M. J.: A Controlled Study of Cold Vaccine. Arch. Otol., 29:704, 1938.

22. Walsh, T. E., and Cannon, P. R.: Immunization of the Upper Respiratory Tract. Arch. Otol., 29:704, 1938.

23. Walsh, T. E., and Cannon, P. R.: Immunization of the Upper Respiratory Tract. J. Immunol., 35:31, 1938.

24. Walsh, T. E., and Cannon, P. R.: Studies on Acquired Immunity in Rabbits to Intranasal Infection with Type I Pneumococcus. J. Immunol., 31:331, 1936.

25. Bull, C. G., and McKee, C. M.: Resistance to Intranasal Infection in the Absence of Demonstrable Antibodies. Am. J. Hygiene, 9:490, 1929.

26. Linton, C. S.: Resistance of the Upper Respiratory Mucosa to Infection. Annals of Otology, Rhinology and Laryngology, 42:64, 1933.

27. Clark, A. R.: The Role of Clasmatocytes in Protection Against the Pneumococcus. Arch. Path., 8:464, 1929.

28. Ward, E.: Prevention of the Common Cold. Lancet, 1:914, 1935.

29. Walsh, T. E., and Cannon, P. R.: Unpublished observations.

LXXII

CORRECTIVE SURGERY OF THE NASAL TIP*

JOHN MARQUIS CONVERSE, M.D.

NEW YORK

Plastic surgery of the external nose during recent years has been greatly improved. This is particularly true in corrective operations upon the nasal bones, the lateral cartilages and the septum. Surgical correction of the tip of the nose is more difficult. It is, however, essential, for deformities of the nasal tip are the most conspicuous of all nasal deformities.

ANATOMY

The tip of the nose is constituted by a cartilaginous framework, the alar cartilages, supported by a central pillar, the septum.

In the midline the two alar cartilages meet, supported by the anterior-superior angle of the quadrangular septal cartilage. The alar cartilages then present a sharp turn downward to embrace the mobile sub-septal cartilage and form the columella.

The portion of each alar cartilage situated laterally to the point where it turns is termed the lateral crus, that situated medially, the medial crus.

This cartilaginous framework is lined on its inner surface by mucous membrane. It is covered on its outer surface by muscles, subcutaneous tissue and skin.

The muscles which cover the alar cartilages and are inserted upon them are muscles of expression, whose nerve supply is derived from the facial nerve. They are the compressor naris, the depressor alae nasi, and the dilator nares, anterior and posterior. Their actions produce dilatation or constriction of the alae and elevation or depression of the tip.

The blood supply of the nasal tip is abundant. The arterial supply is derived from two main sources, the ophthalmic artery and

*Photographs by Mr. James Weeks from the Photographic Laboratory of the Manhattan Eye, Ear and Throat Hospital. Drawings by R. Pierce Thompson and by Sheila Paull.

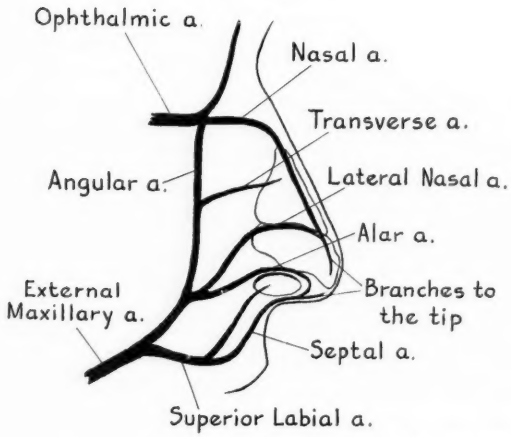


Fig. 1. Drawing illustrating the arteries of the external nose.

the external maxillary artery, and one secondary source, the infra-orbital artery. The arteries anastomose (Fig. 1).

The veins drain into the anterior facial vein. The sensory supply to the nasal tip is derived from branches of the fifth nerve, the naso-ciliary nerve medially, the infra-orbital nerve laterally.

DEVELOPMENT

This is the structure of the nasal tip in the adult. It is however through a long period of development that it is achieved.

Embryologically the alar cartilages are derived from the primitive cartilaginous nasal capsule. This is a well advanced structure by the end of the third month of embryonal life. A greater portion of the cartilaginous capsule becomes ossified to form the nasal bones and various intra-nasal structures. Ossification centers appear during the sixth month of embryonal life. A lesser portion of the cartilaginous capsule persists to form the quadrangular cartilage of the septum, the lateral, and the alar cartilages and the columellar cartilage. According to Schaeffer the original continuity of the nasal capsule is broken up by a division into smaller portions and an ingrowth of connective tissue at certain points. In this way during the sixth month the individual lateral cartilages and the alar cartilages are formed. This explains the presence of supernumerary cartilages in the alae.

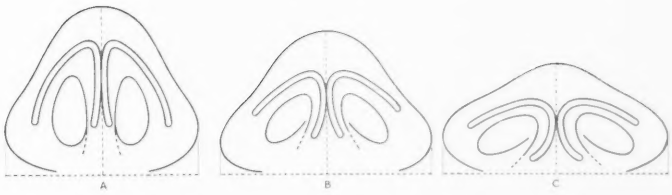


Fig. 2. The base of the nose: *A.* In the white race; *B.* in the yellow race; *C.* in the negroid race.

During early childhood the external nose is very different in shape from the adult nose. It is flat and smaller in proportion to the dimensions of the face than the adult nose. The alar cartilages are extremely small. For this reason it is essential that in cases of harelip which present an asymmetry of the nasal tip due to a disproportion of the alar cartilages, no correction be attempted until the nose is fully developed.

Around the age of six years the alar cartilages are well defined small structures which can be outlined under the mucosa of the nose by retracting the ala and palpating its intra-nasal surface with a small probe.

At puberty the whole of the external nose increases rapidly in size along with the septum. At this time the alar cartilages begin to assume their definitive shape and proportions.

ARTISTIC ANATOMY

The shape of the nasal tip is that of the alar cartilages, for the muscles and the skin merely follow the form of the cartilaginous framework. Whatever variation may exist in the shape of these cartilages reveals itself in the external shape of the tip and also in the shape of the nostrils. For example rounded alar cartilages give a rounded tip and round nostrils.

In a general way it is accepted that the proportions between the height and the width of the base of the nose are those illustrated (Fig. 2A). This differs in Asiatics (Fig. 2B) and in negroes (Fig. 2C).

It would be fallacious however to reduce artistic anatomy to geometric expressions. Great variations exist within normal limits,

depending greatly upon the shape of the whole nose, that of the face in general and even facial expression. In evaluating variations from the normal and their eventual correction a great deal depends upon the surgeon's artistic judgment.

ANATOMICAL DIAGNOSIS OF THE DEFORMITY

The shape of the alar cartilages is extremely variable. The variations observed in over one hundred operations for tip corrections apply to both the size and the shape of the alar cartilages. The thickness of the cartilages varies from one mm. to four mm. Patients with wide hypertrophic tips present thicker alar cartilages (Fig. 8) than those having thin tips with collapsing alae (Fig. 11). The alar cartilages extend further laterally into the ala in the thin type of nose than in the broad type. In the former they may occupy three-quarters of the length of the ala (Fig. 11); in the latter they may occupy only the medial third of the ala, flaring out nearly horizontally and protruding under the skin, thus producing a large square tip (Fig. 8). At times there is a generalized hypertrophy of the alar cartilages in every direction giving a large bulbous tip (Fig. 18). In some cases there is a congenital absence of most of the lateral crus of the alar cartilage. The cartilage is fully developed in its medial portion but ends very near the midline thus producing a peculiar square tip with collapsed alae (Fig. 12). Generally a little over one cm. in width the alar cartilage may assume great proportions, overlapping the lateral cartilages above and constituting most of the cartilaginous external nose.

The medial crura may be hypertrophic and produce a widening of the columella which may be sufficient to cause nasal obstruction (Fig. 11). The shape of the alar cartilages is varied. In the thin nose with a pointed tip the alar cartilages take a sharp bend downwards (Fig. 11). In the broad nose with a rounded or square tip the cartilages flare out in a wide curve (Fig. 8). The alar cartilages may not meet at the tip, thus leaving an interstice and giving the appearance of a bifid nose.

Following trauma due to crushing of the anterior-superior portion of the septum the cartilages lack support. They present a rounded contour producing a rounded tip with round nostrils (Figs. 4 and 6).

In deviations of the nasal tip due to a deflection of the anterior portion of the septum especially following trauma, each cartilage has a different shape. When the trauma has occurred in childhood, with

the growth of the nose a marked disproportion develops. The alar cartilage opposite to the deviated side is longer (Fig. 14). This condition is particularly marked in the nasal deformity accompanying harelip.

SURGICAL APPROACH TO THE NASAL TIP

Having made an anatomical diagnosis of the deformity the question arises how the cartilages of the nasal tip can be approached. Although various procedures have been used for tip correction, to Kazanjian goes the credit of having first devised a method for complete reconstruction of the nasal tip. To be satisfactory such an approach must present two essentials: adequate exposure of the cartilages and preservation of the physiology of the region.

Adequate exposure of the nasal tip can be obtained by two methods:

A. Tip exposure "from above". In this first method (Fig. 16) complete subperichondrial dissection of each alar cartilage is carried out after the exposure of the lower border of the cartilage through an incision near the free border of the nostril. This incision is made along the full length of the cartilage and is carried medially following the curve of the cartilage down the columella in front of the anterior border of the septum. Following this preparation the whole of the cartilaginous tip may be drawn out through one side or the other (Fig. 16).

B. Tip exposure "from below". In this method (Fig. 17) an incision about one cm. in length is made along the anterior border of the alar cartilage. A second incision is made also one cm. along the superior border of the alar cartilage just below the inferior border of the lateral cartilage. The mucous membrane lining the alar cartilage is then elevated along the whole surface of the alar cartilage as far laterally as its free lateral border.

A subperichondrial elevation of the muscle layer covering the superior surface of the alar cartilage is performed so that the cartilage is completely separated from the surrounding tissue. The alar cartilage can be seized with a fine hook and drawn out through the vestibular incision (Fig. 17).

Both methods avoid any disturbance of the physiology of the nose. The muscles, nerve and vascular supply are undamaged. The mucous membrane is intact. Injury to the muscles or their nerve supply would result in an expressionless nose. Following such an

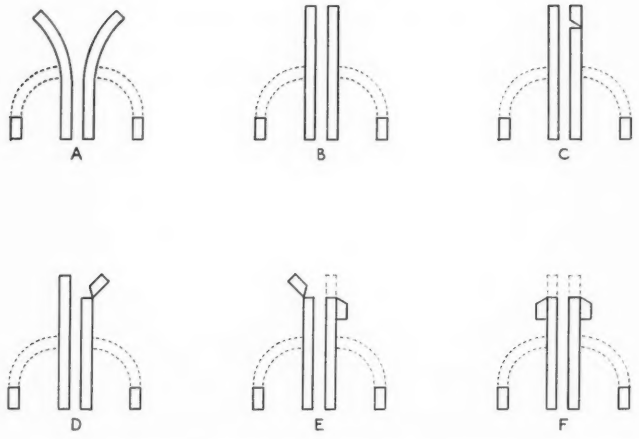


Fig. 3. Shows various steps of "up and down" operation. The folding down of the upper portion of the raised alar cartilage gives good contour to the newly raised tip. This procedure permits the removal of a large amount of cartilage from the lateral portion of the alar cartilages.

operation the patient should be able to dilate the nostrils and to elevate the tip. Destruction of mucous membrane would result in disastrous nasal stenosis. All incisions should be carefully sutured with fine silk so as to obtain rapid healing and minimal scarring.

CORRECTIVE SURGICAL METHODS

Increase in transverse dimensions. The large bulbous type of nasal tip is due to an overdevelopment of the alar cartilages. The excess of cartilage is removed at the point of the maximal flare by a careful subperichondrial dissection with fine sharp scissors. The piece of cartilage removed is generally triangular with its base anteriorly. In the average nose this may be done by using the method of exposing the cartilage "from below"; in the more marked cases it is preferable to use the exposure "from above." In the extreme cases it may be necessary to resect as much as one-half of each alar cartilage.

The flattened or rounded nasal tip (Figs. 4 and 6) is generally the result of trauma and is often found along with saddle nose deformities. Correction must consist in a narrowing and an elevation of the nasal tip. The author has modified Kazanjian's original oper-

ation. Using the exposure "from above" the whole of the cartilaginous tip is exposed and extruded (Fig. 16). The operation is then carried out as illustrated (Figs. 3, 4 and 5). This "up and down" operation permits the displacement of cartilage from where there is too much of it to the midline where it is deficient. In cases associated with a saddle-back deformity of the bridge the approach "from above" may be used not only to resect the excess alar cartilage but also as a route from the insertion of the graft (Figs. 6 and 7). The author uses rib cartilage as a rule; in the more severe types, ileal bone. When ileal bone is used the graft is supported at the tip by a columellar strut.

Increase in the vertical dimensions of the nasal tip. Such a case is illustrated (Fig. 10). Correction is achieved by means of the tip exposure "from below" (Fig. 17). A quadrangular piece of cartilage is excised from each alar cartilage and also from the septum, thus diminishing the vertical dimensions (Figs. 10 and 11). The operation is completed by excision of the medial crura of the alar cartilages in the columella.

A peculiar condition is illustrated (Fig. 12). Here there is a congenital absence of all the lateral portion of each alar cartilage. This results in an alar collapse and further accentuates the breadth of the tip. Using the tip exposure "from above" the upper lateral cartilages were excised and transplanted into the ala after remodeling the tip proper (Figs. 12 and 13).

Deviations of the nasal tip. In tip deviations, as in all nasal deviations, the correction of the deviated portion of the septum is the *sine qua non* of a successful result. The whole cartilaginous tip is then exposed "from above", and the tip may be extruded and examined under direct vision (Fig. 16). The asymmetry must then be corrected. There is a disproportion in the length of the alar cartilages. The alar cartilage on the side opposite to the deviation is longer, and there is a flattening of the whole ala (Fig. 14).

The excessive length of the cartilage is corrected by a resection of the lateral portion of the cartilage, and in order to correct the flattening of the ala this piece of cartilage is laid over the remaining alar cartilage and sutured in position (Fig. 15).

A series of incisions are made through the cartilages of the tip to break their spring and make it possible to suture them together in a symmetrical fashion (Fig. 15).

This type of operation may be used to correct the nasal deformities secondary to harelip. These cases, however, constitute a definite type that should be considered at more length in a special article.

SUMMARY

Following a review of the anatomy and the development of the nasal tip, deformities of this region are considered, various methods for their surgical correction are described, and the results obtained are illustrated.

121 E. 60TH.

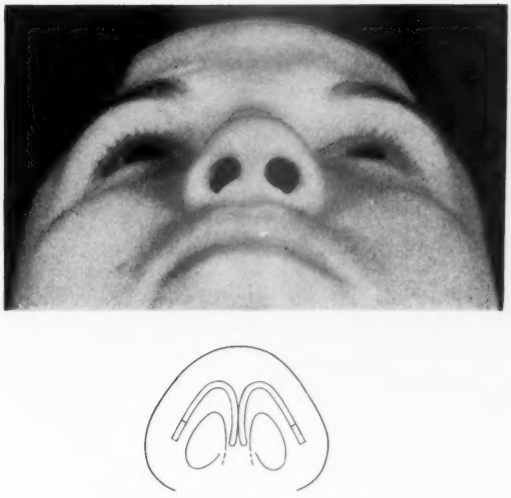


Fig. 4. Tip presenting rounded contour following trauma. Note rounded nares.

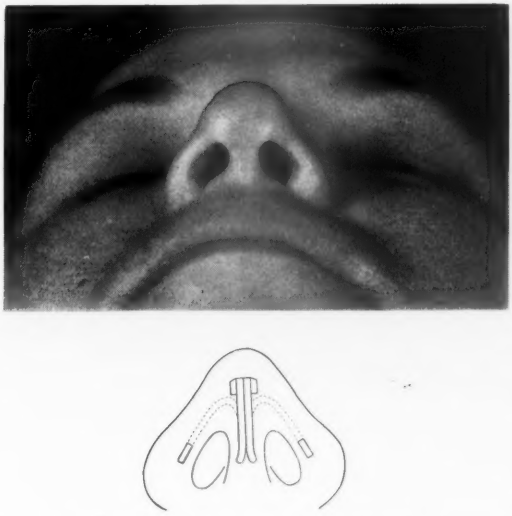


Fig. 5. Result following "up and down" tip raising operation. Note change in shape of nares.

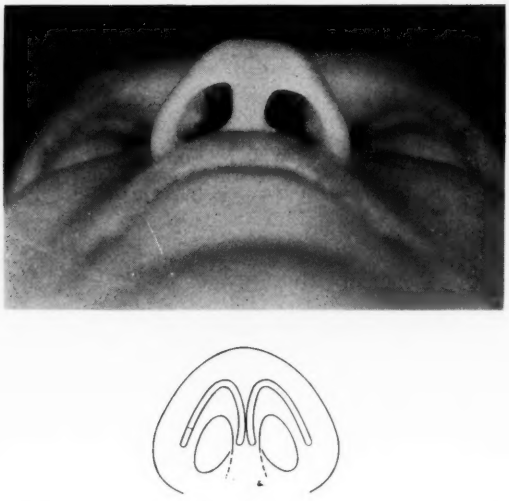


Fig. 6. Post-traumatic flattening of tip accompanying saddle nose.

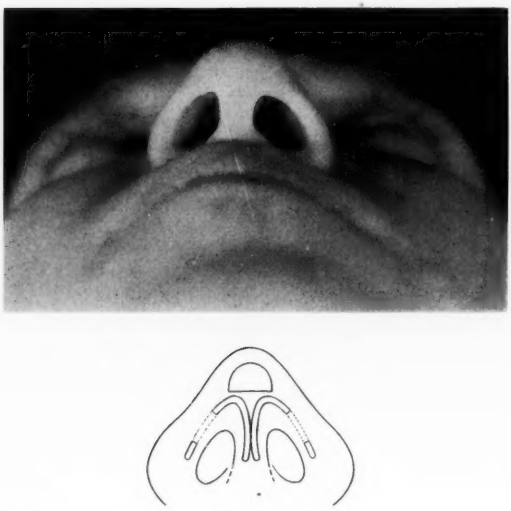


Fig. 7. Appearance of tip after correction following insertion of a graft and remodeling of alar cartilages.

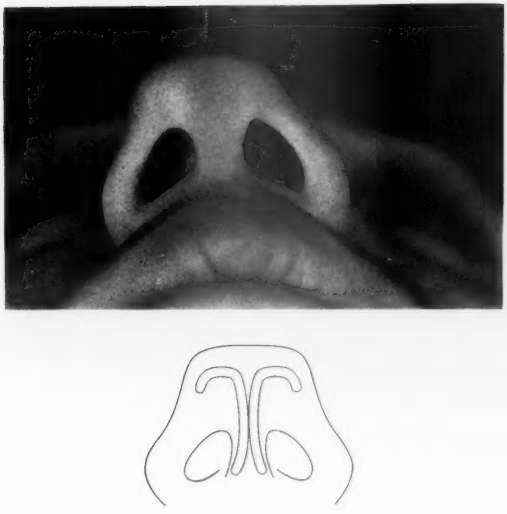


Fig. 8. Large hypertrophic tip.

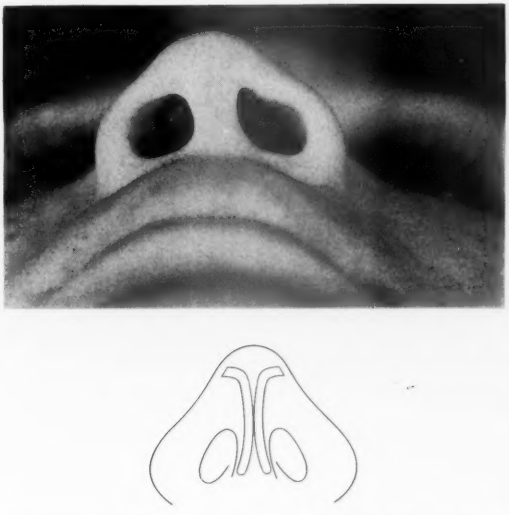


Fig. 9. Appearance after operative remodeling and excision of the lateral portions of each alar cartilage.

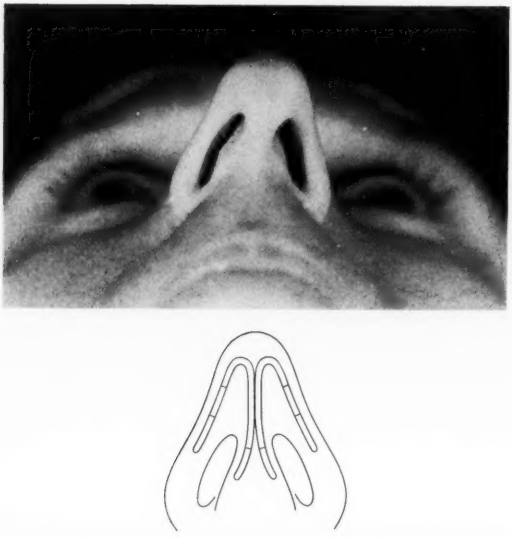


Fig. 10. Extremely elongated nasal tip; wide columella due to hypertrophic medial crura of the alar cartilages.

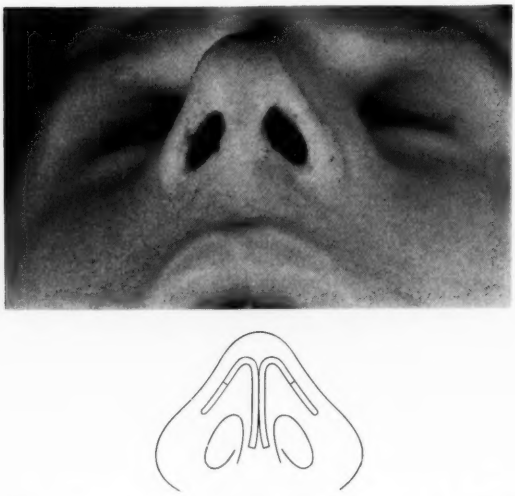


Fig. 11. Appearance after resection of cartilage from the alae and from the medial crura of the alar cartilages.

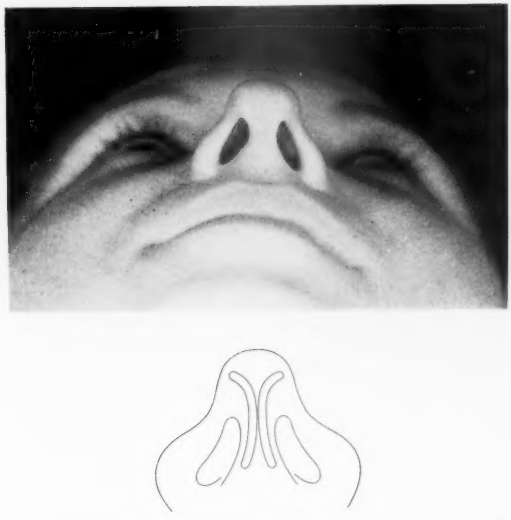


Fig. 12. Congenital absence of the lateral portion of the alar cartilages with resultant alar collapse.

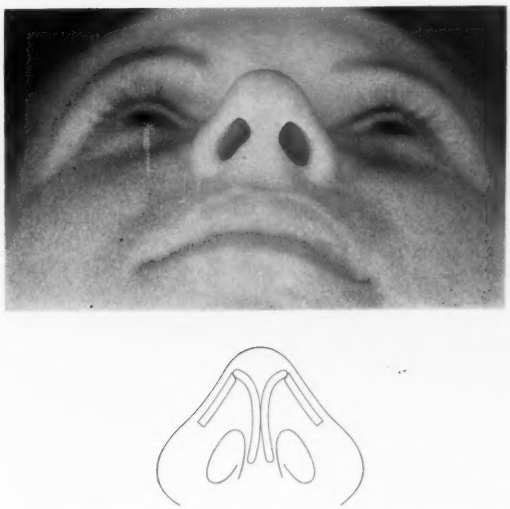
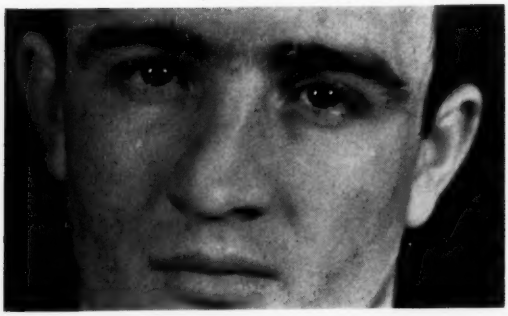
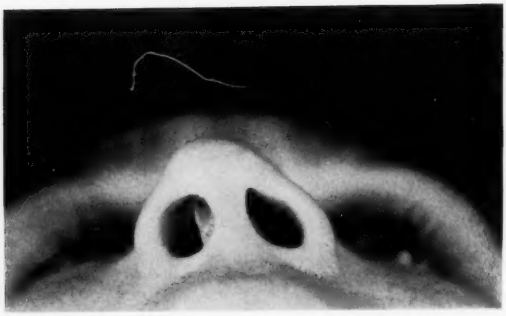


Fig. 13. Appearance after cartilage grafts and tip remodeling.





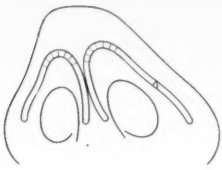


Fig. 14. Asymmetrical, deviated tip.

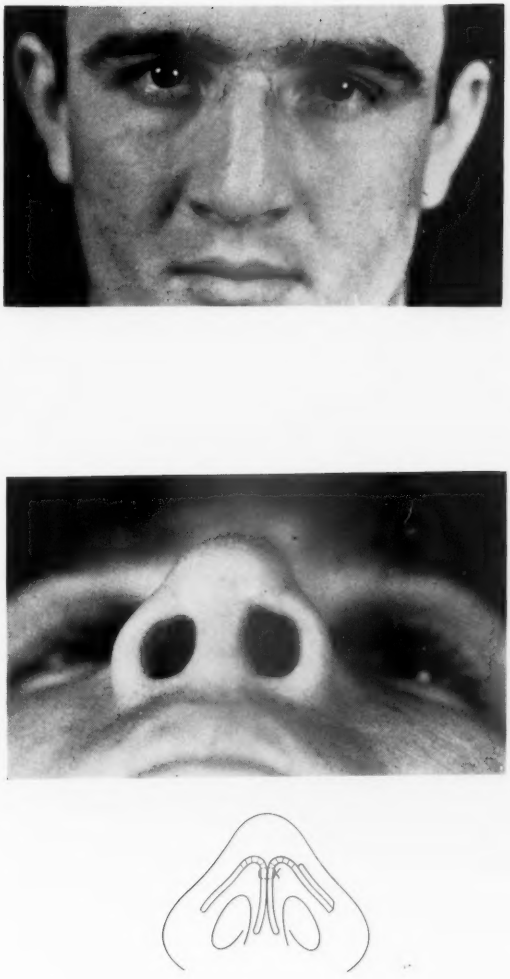


Fig. 15. The case shown in Fig. 14, after surgical correction.

Fig. 16. Photograph showing the exposure of the cartilages of the tip "from above." Both alar cartilages are shown at their point of junction with the tip. They have been extruded and can be seen under direct vision.

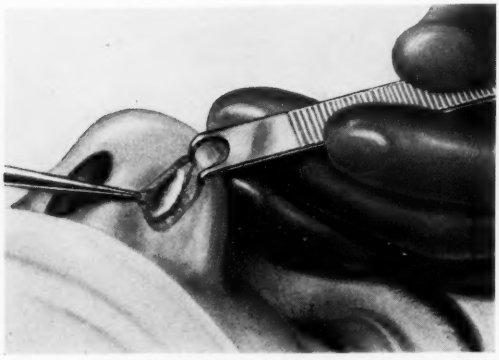


Fig. 17. Drawing representing exposure of the alar cartilages obtained by the exposure "from below."

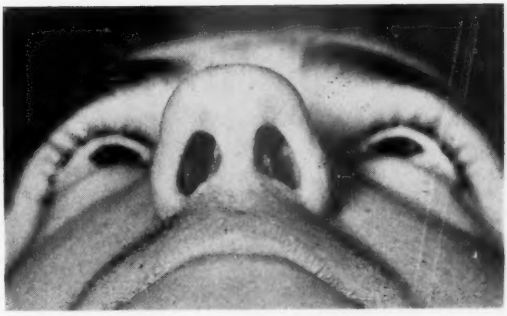


Fig. 18. Large tip; hypertrophic alar cartilages.

Fig. 19. Result obtained by partial resection of alar cartilages and tip remodeling.

LXXIII

NASAL ATRESIAS: A SURGICAL CRITIQUE

ALBERT A. CINELLI, M.D.

NEW YORK

ATRESIA OF THE ANTERIOR NARES

Atresia of the anterior nares is a very rare condition. It may be congenital or acquired, complete or incomplete, unilateral or bilateral.

Etiology: Various theories are advanced to explain its congenital origin. Schaeffer states that in many instances the anterior nares become secondarily obstructed by marked proliferation of the epithelium. Plugging of the nares is a common condition from the fortieth to the sixtieth day of embryonic life. In the majority of cases there is a solution of the atretic plug, the result of central degeneration and actual shedding of the epithelium. One often finds shreds of epithelial masses protruding from the nares of fetuses. Thomson merely asserts that this condition is caused by a "web of skin" at the junction of the vestibule with the nasal chamber proper. Cheatle suggests that the closure may be due to an inflammatory process during intra-uterine life. Williams mentions syphilis and trauma as causes, and Kelemen reports a case of anterior occlusion of both fossae due to developmental anomaly (pouch of Jacobson's organ).

Acquired atresia of the anterior nares is usually due to trauma or disease. Foreign bodies (bullets, pins, etc.), faulty submucous resection and incorrect plastic surgery on the tip have been found to produce atresia of the nasal passages. Dewey reports a case of atresia of the anterior nares complicating variola. Washburne reports its occurrence due to syphilis in two sisters.

Often atresia of the nares goes unnoticed, particularly in the new-born. In these cases the infant has difficulty in suckling; the face turns blue during prolonged nursing. Even feeding with a spoon or a dropper may become impossible in these cases. Asphyxia neonatorum is sometimes due to this undiagnosed condition. Bolzano cites several cases of slow asphyxia due to complete occlusion of the nares.

Even when the atresia is not so serious as to jeopardize life, certain physical malformations may develop. Darcissac describes a lat-

eral expansion of the upper jaw in a case of marked atresia in a patient 30 years old. Aprile points out the influence of nasal stenosis on the development of the maxillary sinus. In some instances pulmonary tuberculosis, rickets and emaciation may be the result of atresia of the nares.

Therapy: Various methods have been recommended for the correction of this malformation, among them electrocoagulation, incision and packing, and excision of atretic membrane with insertion of a rubber tube. These methods have proved futile for the most part because contraction of the resulting granulation tissue causes shrinkage of the nasal opening. However Joseph, Kirschner, Dewey, O'Connor and Blair have prevented contraction and obtained successful results by using a Thiersch graft to line the raw area of the nares after excision of the atretic membrane.

The technique of repair is the same for complete or incomplete atresia. It is best done, with a minimum of bleeding, under local anesthesia. Dissection of the entire atretic web is essential. In order to have a rigid background for implantation of the coming graft, it is necessary to remove every vestige of pathology, exposing the alar cartilage with its crura, the upper lateral cartilage, the apertura piriformis and the septum. When this is done a large raw area presents itself. It is absolutely necessary to give this an epithelial covering; otherwise granulation tissue followed by cicatricial tissue will produce a greater deformity than the original condition. Blair's split graft is our precedure of choice.

An over-impression of the denuded area is made with a soft rubber tube. The split graft is placed around this tube, raw surface exposed, and its edges are sutured. It is then placed *in situ*. It is essential that this graft be well immobilized; otherwise it will not take. Another important point to remember is that there must be some over-correction to allow for a certain amount of contraction which is inevitable in spite of all precautions. The softness of the tube will prevent any possible pressure necrosis.

The nose is kept clean postoperatively by passing a small rubber catheter through the tube and removing all secretion and debris by dry suction. This is followed by instillation of metaphen and neosynephrin for antisepsis and aeration.

The tube is removed in ten days, and the area is inspected and cleaned. A new epithelial lining of the nose should now be presented.

The newly created cavity must be further treated with dilatation and immobilization; otherwise there will be great shrinkage and contraction. The same tube cleaned, or another like it completely filling the new spaces, should be reinserted for at least another ten days.

When the support is removed at the end of this interval, all granulating tissue should be coagulated. A certain amount of contraction will eventually take place, but this has been provided for by the over-correction.

CHOANAL ATRESIA

Otto, in 1829, was the first to discover a congenital obstruction of the posterior nares, quite accidentally, while performing an autopsy. It was not until 1853 that the first surgical case was reported by Emmert, in a boy seven years old. The obstructing mass was eradicated with the use of a curved trocar, and this technique prevailed until 1885 when von Schroetter reported the successful use of electrocauterization in choanal atresia. This was immediately followed by the employment of many different chemical caustics.

Uffenorde, in 1908, was the first to advocate surgical removal of the choanal mass by means of chisel and curette, through the nasal septal route. This method was soon discarded.

Katz in 1913 and White in 1919 advocated the technique of removing the obstructing wall first and then taking away the posterior part of the vomer. This is perhaps the method of choice today.

Bourgeois and Leroux, quoted by Imperatori and Burman in 1922, initiated the successful eradication of choanal atresia by electrocoagulation. This technique has not been very popular and very little attention has been given to this approach. In 1940, Morgenstern reported the successful treatment of a case by this method.

Blair, in 1931, was the first to introduce skin grafting. He used a large split graft sutured around a tube, with the raw surface of the graft coming in direct contact with the raw surface of the choanal region after eradication of the obstruction. In 1938 Donnelly reported a case of right-sided choanal atresia corrected by the use of a full thickness graft sutured around a No. 18 French woven catheter.

Statistical data reveals about 220 cases reported to date, the majority in the past twenty years. Diagnosis of this condition is relatively simple because of the many physical means at our disposal to ascertain the pathology. Treatment appears to present a more

difficult problem, chiefly because the individual surgeon is rarely confronted with these cases. When he does encounter one and refers to the literature, he is likely to be confused by the diversified procedures advocated.

If we bear in mind the underlying pathology, it is not difficult to adopt suitable corrective measures. In the unilateral type of atresia, operation may not be necessary. Most often these cases are recognized by chance upon routine examinations without the patient's pre-knowledge of the condition. Surgical correction depends upon the underlying pathology, as will be seen in the bilateral type.

Another important question concerns the opportune time to operate. Correction has been attempted in these congenital atresias at all ages, from the early days of infancy to adult life.

In the bilateral congenital type, pediatric care in connection with the problem of infant feeding is far more important than surgical intervention. It is best to wait until the infant has developed some resistance for the operation. Two years of careful nursing should be adequate before surgical intervention.

This type of operation is governed by the nature of the atresia. If the choanal atresia is osseous, mere perforation of the mass to create an opening is usually insufficient. Many ingenious devices have been utilized to maintain this postoperative patency, such as tubes, obturators, catheters, post-nasal tampons, impregnated medicated gauze strips, etc. However the surgically created aperture usually closes after several months because of the stubborn nature of the osseous occluding growth. Blair's technique of skin-grafting this type of atresia is unquestionably the procedure of choice.

The important fact to remember is that the field must be absolutely dry before applying the skin graft. Even a small blood clot is sufficient to cause failure. The opening should be perfectly smooth so that the graft is snugly adherent to the exposed raw surfaces.

Another important requirement is that the graft should have complete immobilization; otherwise it is certain to slough. The graft is kept in place on a rubber tube or French catheter for at least 10 days before the support is removed. A split graft is more certain to take than full thickness graft.

Almost any instrument can be used to remove enough of the mass for the implantation of the graft. Most commonly employed are the trocar, chisel, hammer, curette, dental burr and drill.

In removing any atresia it is most important not to injure the mucosa of the nasopharynx. This can easily be prevented by inserting a choanal plug, or if necessary the left index finger, into the nasopharynx.

The anesthesia is the same for all atresias. Chloroform is best for children, while intra-tracheal anesthesia has proven ideal for adults.

In the membranous form of choanal atresia good results are obtained by removing surgically a large portion of the obstruction and the posterior third of the septum and following this at a later date with electrocoagulation for future granulations.

A sphenoid punch forceps should be sufficient to remove a large portion of the atresia. The posterior third of the septum, including the vomer and the lower section of the perpendicular plate of the ethmoid, is then removed in order to give plenty of room. A choanal plug impregnated with balsam of Peru and castor oil (as in posterior nasal bleeding) is snugly inserted into the large created opening and fastened securely to the auricle. The packing is removed on the fifth day and reinserted. This choanal plug seldom causes any otitic complications. It is easy to manipulate and not too annoying to the patient.

On the ninth or tenth day, when the second packing is removed, the created space is thoroughly cleaned with sprays and irrigations. Under strong illumination all granulating surfaces are then coagulated as in tonsillar remnants. This coagulation is repeated once a week; in the interim the area is kept clean with sprays and irrigations. Some contraction will take place, but there is usually enough of an opening for sufficient aeration and drainage.

In removing a portion of the atresia, whether osseous or membranous, it is best not to go too far laterally as there is some danger of injury to the nerves and blood vessels coming through the pterygopalatine and posterior palatine canals.

ATRESIA OF THE NASOPHARYNX

This condition is not common. There is a scarcity of material in the literature about it. Wright and Smith collected about 69 cases and advocated the use of monochloracetic acid on the raw surface after division of the atresia. They concluded that the congenital type is exceedingly rare. The acquired form is usually due to syph-

ilis, trauma, diphtheria and tuberculosis. From its etiologic basis, one can readily understand why this pathology is a rarity today.

Symptoms depend upon the extent of the atresia, which is seldom complete. There is usually difficulty in nasal breathing. Obstruction of the eustachian tubes gradually causes loss of hearing. The voice develops a pronounced nasal tone, and interference with proper drainage from the sinuses leads to profuse, foul, greenish purulent secretions in the nose, which are most annoying. There is usually some loss of smell and taste associated with this condition.

The congenital type of nasopharyngeal atresia may not be easy to diagnose. As a rule the patient comes for the usual adenoid obstruction. It is at operation, when the finger experiences a new sensation, that the atresia is usually recognized. A smooth membrane is found to extend from the posterior aspect of the soft palate usually high up to the posterior nasopharyngeal wall. It is yielding to the touch, and the vault is felt to be very narrow.

This congenital membrane or diaphragm can easily be divided with the index finger, aided if necessary by a probe passed into the nose down into the nasopharynx and forcing its way through the membranous obstruction. It is usually sufficient to paint the raw surface with monochloracetic acid.

This type of atresia seldom recurs. In more stubborn cases it may be necessary to pass a catheter bougie infrequently.

It is the acquired form that presents the problem of retaining patency. The outcome depends upon two factors: the nature of the pathology and the extent of the involvement.

Except when the acquired atresia is of luetic origin, the prognosis for the repair is good. In syphilis, tissue has a tendency to break down and cause destruction of other tissue. When arrested, this tendency yields to marked cicatrization with contraction. Nonluetic scar tissue is free from this undesirable characteristic.

MacKenty, in 1927, came forward with an ingenious surgical method which was the first to prove efficacious in this type of case. His operation was based on the principle of Nichols that the defect has its origin in a tendency of wounds to heal from their periphery toward the center. For MacKenty's flap operation to be successful, it is essential that the mucous membrane on the posterior pharyngeal wall below the normal line of the velum palati should not be entirely destroyed. The condition is almost hopeless if there is nothing but

scar tissue; if the atresia above is extensive and the whole pharynx is contracted to the center; and especially if syphilis is the cause. A good result may be expected if the atresia is shallow, if the mucous membrane below it is partly preserved, if the pharynx has a fairly wide and normal look and if the condition is traumatic or due to a simple inflammation. Dilatation should be carried out after the operation to such an extent and for such time as seems indicated.

When the pathology is widely infiltrated and especially if syphilis is the cause, the problem of repair is indeed most difficult.

McIndoe bores a hole between the adherent palate and the posterior pharyngeal wall from the pharynx into the nasal cavity. He then places a mould or dental stent covered with a Thiersch graft between these two separated raw surfaces. The mould is retained by a double silk thread tied around the columella and retained in place for at least two or three months. In this fashion he reports that an excellent passage is created. Drainage of the nasal cavity and sinuses is attended by remarkable improvement in the patient's general health.

The essayist had one case in which this technique was tried, but unfortunately the graft did not take, and the end result was unsatisfactory.

Many plastic operations such as incision with dilatation or cauterization have been advanced to correct this difficult problem. Some successful cases have been reported with the use of diathermy. Others have reported using the flaps from the mucous membrane of the adjacent cheeks. These, like others, fell by the wayside.

Figi reported 18 cases of stenosis of the nasopharynx from the Mayo Clinic. Four cases were the result of faulty tonsillectomies and adenoidectomies, and two cases were due to faulty tonsillectomies alone; three cases were due to hereditary syphilis, two cases to acquired syphilis, two cases to indeterminate inflammatory processes, two cases to rhinoscleroma, one case to diphtheria, one case to sulphuric acid and one case to congenital abnormality.

Figi, like MacKenty, followed the principle of Nichols. Nichols treated 13 cases successfully by inserting a seton at the base of the web until cicatrization took place, and then incising to this point he inserted a silk suture through the small nasopharyngeal opening well into the lateral extent of the region of scarring. This suture was tied and left in place until a cicatrized tract developed. The pos-

terior border of the soft palate was then freed from its attachment to the pharyngeal wall out to this point.

In Figi's last six cases, which were successful, he used the same technique with some variations. He added a small lead weight, which was clamped on over the ends of the suture, and allowed the suture to cut through by itself. This usually required one to two weeks. When this happened, it was replaced by another, taking a wider lateral bite. Subsequent dilatation with rubber-tipped Kelly forceps was employed in most cases.

Recently the writer employed a more simplified technique, not original, which proved successful. The scarred soft palate was gently released from its attachment to the posterior pharyngeal wall by a transverse incision. Palatal scarred tissue was removed until a large opening was made. A very large but short soft rubber tube with hinges on both sides was introduced via the mouth. The hinges rested on the posterior surface of the lateral walls of the soft palate. The upper end of the tube faced the choana, and the lower end was seen slightly coming through the oropharynx. In this fashion the tube was well immobilized and unable of its own account to fall either into the mouth or forward into the nasal chambers.

The tube was kept in that position until all the margins were completely epithelialized. It can be easily retained for several months without causing any undue damage. There are some discomforts for the first few days until the patient becomes accustomed to it.

Soft rubber tubes do not produce an irritation as do the hard ones and perhaps because of this prove to be most useful in these cases.

When surgery is completed, these patients should receive vocal training. The palatal muscles are essential for phonation, and having been unable to function because of the atresia, they require re-education after operation.

1021 Park Avenue.

BIBLIOGRAPHY

1. Aprile, V.: Influence of Stenosis on Development of Maxillary Sinus. Exper. Research, Valsalva, 10:48 (Jan), 1934.

2. Balzano, I.: Slow Asphyxia Due to Complete Occlusion of Nasal Fossae. Oto-Rhino. Laryng. Ital., 3:404 (July), 1933.

3. Blair, V. P.: Congenital Atresia or Obstruction of the Nasal Air Passages. Annals of Otology, Rhinology and Laryngology, 40:1021 (Dec.), 1931.

4. Cheatle, A. H.: Proct. Laryng. Soc., London, 9:98 (April), 1902.

5. Darcissac, M.: Lateral Expansion of Upper Jaw in Case of Marked Atresia in Patient 30 Years Old. Bull. et Mem. Soc. d. Chirurgiens de Paris, 25:296 (May), 1933.

6. Dewey, C. H.: Atresia of Nares as Complication of Variola. Case Report. Military Surgeon, 65:702 (Nov.), 1929.

7. Joseph, Jacques: Nasenplastik und Sontige Gesichtzplastik. Text, Leipzig, 1928-31.

8. Kelemen, G.: Anterior Occlusion of Both Fossae Due to Developmental Anomaly (Pouch of Jacobson's Organ). Budapesti orvosi ujsag, 31:101.

9. Kirschner, M.: Allgemeine und spezielle chirurgische Operationslehre. Page 470. Verlag Von Julius Springer, Berlin, 1935.

10. O'Connor, G. B.: An Operation for the Correction of Atresia or Stenosis of the Anterior Nares. Arch. Otolaryng., 25:208 (Feb.), 1937.

11. Schaeffer, J. P.: Various Types of Congenital Atresia with Their Genetic Interpretation. Tr. Am. Laryng. Assoc., 56:126, 1934.

12. Thomson, St. Clair: Diseases of the Nose and Throat. Page 127, Cassell & Co., New York, 1926.

13. Washburne, A. C.: Congenital Atresia of Anterior Nares. Two Cases in Sisters. Arch. of Otolaryng., 16:789 (Dec.), 1932.

14. Williams, P. W.: Proc. Laryng. Soc., London, 4:32 (Dec.), 1895.

15. Asherson, N.: Bilateral Congenital Occlusion of Posterior Choanae Due to Membranous Diaphragm. J. Laryng. & Otol., 45:344, 1930.

16. Binnerts: Arch. f. Laryngol. u. Rhinol, 34:324, 1921.

17. Blair, V. P.: Congenital Atresia or Obstruction of the Nasal Air Passages. Annals of Otology, Rhinology and Laryngology (Dec.), 1931.

18. Boulay, M.: Etude clinique sur L'occlusion congenitale de l'orifice posterieur des fosses nasales. Arch. de med. d. enf., 140:156, 1902.

19. Bourgeois and Leroux: Imperatori and Burman: Diseases of Nose and Throat. Lippincott & Co., Philadelphia, page 638, 1935.

20. Braun and Clasen: Cited in Skillern: Accessory Sinuses of the Nose. J. B. Lippincott Co., Philadelphia, 1913.

21. Brady, A. J.: J. Laryngol., Rhinol. & Otol., 33:49, 1918.

22. Colver, B. M.: Congenital Choanal Atresia: Two Cases of Complete Bilateral Obstruction. Annals of Otology, Rhinology and Laryngology, 46:358-375 (June), 1937. Has review of literature and bibliography.

23. Charousek, G.: Arch. f. Ohren-Nasen- u. Kehlkopf., 110:209, 1922.

24. Corwin, T. W.: The Nasal Septum as a Factor in Stenosis. J. M. Soc. New Jersey, 26:201-206 (March), 1929.

25. Cohen, J. S.: Diseases of the Throat and Nasal Passages. Second Ed., N. Y., 1880.

26. Dean, L. W.: The Nose, Nasopharynx and Paranasal Sinuses. Practice of Pediatrics, 2:3, 1936.

Paranasal Sinus Disease in Infants and Young Children. J. A. M. A., 85:317 (Aug.), 1925.

27. Dewey, C. H.: Atresia of Nares as Complication of Variola: Case Report. Mil. Surgeon, 65:702 (Nov.), 1929.

28. Donnelly, J. C.: New Method of Operation for Congenital Atresia of Post Nares. Arch. of Otolaryng., 28:112 (July), 1938.

29. Emmert, C. F.: Lehrbuch der Chirurgie Stuttgart. F. & R. Donn., 1853.

30. Fishbein, J. N.: Report of a Case of Choanal Atresia. Rhode Island Med. J., 14:180 (Nov.), 1931.

31. Fraser, J. S.: Congenital Atresia of the Choanae. Brit. Med. J., 2:601 (Nov.), 1910.

32. Garretson, W. T.: Congenital Occlusion of Choanae, with Report of Two Cases. Laryngoscope, 37:263 (April), 1927.

33. Geiger, C. W., and Roth, J. H.: Osseous Choanal Obstruction and Adenoid Tissue. Arch. Otolaryng., 15:441 (March), 1932.

34. Grove, W. E.: Choanal Atresia and Sinus Infection. Arch. Otolaryng., 6:237 (Sept.), 1927.

35. Grove, W. E.: Congenital Atresia of the Right Posterior Nares. Virginia Med. Monthly, 60:682 (Feb.), 1934.

36 Haag, H.: Ueber Gesichtesschadelform, Aetiologie und Therapie der angeborenen Choanalatresia. Berlin, 1899.

37. Hart, V. K., and Peeler, C. N.: Surgical Management of Bilateral Congenital Occlusion of Posterior Nares: Case Report. South. Med. J., 22:1087 (Dec.), 1929.

38. His, W.: Anatomie menschlicher Embryonen. III zur Geschichte der Organe. Leipzig, 1885.

39. Hochstetter, F.: Ueber die Bildung der inneren Nasengange oder primitiven Choanen. Verhandl. d. anat. gesellsch. Jena, 145:151, 1891.

40. Jaques, P.: Rev. de Laryngol., 29:607, 1921.

41. Katz, L., Preysing, H., and Blumenfeld, F.: Handbuch der speziellen chirurgie des Ohres und der oberen Luftwege. Wurzburg, Curt Kabitzach, 3:432, 1911-1913.

42. Kearny, H. L.: Congenital Bony Atresia of the Right Posterior Naris. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 45:583 (June), 1936.

43. Keith, Arthur: Human Embryology and Morphology, 4:159, Edward Arnold, London, 1921.

44. Kirby, J. C.: Laryngoscope, 31:701, 1921.

45. Lemere, H. B. Persistent Bucconasal Membrane in the New-Born. J. A. M. A., 109:347 (July), 1937.

46. Lewy: Cited by K. Vogel: Ztschr. f. Hals-Nasen- u. Ohrenh., 11:121, 1925.

47. Loeb, H.: Operative Surgery of Nose, Throat and Ear. C. V. Mosby Company, St. Louis, 2:116, 1917.

48. Luschka, H.: Ueber angeborene Atresie der Choanen. Arch. f. path. Anat., Berlin, 18:168, 1860.

49. MacKenty, John Edmund: Congenital Occlusion of the Choanae with Report of Two Cases. Med. Rec., 72:387 (Sept.), 1907.

50. Mahoney, B. L.: Case of Choanal Atresia. Laryngoscope, 37:920 (Dec.), 1927.

51. Moncrieff, A.: Nasal Obstruction in the Newborn. Brit. Med. J., 1:1295 (June), 1936.

52. Morgenstern, David: Congenital Atresia of Post-Nasal Orifices. Arch. Otolaryng., 31:653 (April), 1940.

53. McNaught, H. Y.: Congenital Occlusion of the Choanae of the Nose. California State J. Med., 16:193, 1918.

54. Nager, R. T.: Cor. Bl. f. Schweizr. Aerzte 45:1339, 1915.

55. Otto, A. W.: A Compendium of Human and Complicative Anatomy. Translated by J. F. South. B. Fellowes, London, 1831.

56. Porter, W. G.: Notes on a Case of Congenital Atresia of Choana. Edinb. Med. Jour., 129:133 (Feb.), 1906.

57. Ronaldson, T. R.: Note on a Case of Congenital Closure of the Posterior Nares. Edinb. Med. Jour., 20:1035, 1880.

58. Schaeffer, J. D.: Atresia of the Choanas: Its Incidence and Cause. Laryngoscope, 43:480 (June), 1933. (General, no case report.)

The Various Types of Congenital Atresias of the Nose and their Genetic Interpretation. Tr. Am. Laryng. Assoc., 56:126, 1934.

59. Shea, J. J.: Nasal Obstruction and Systematic Consequence. Tr. Am. Acad. Ophth., 36:199, 1931.

60. Stewart, J. P.: Congenital Atresia of Posterior Nares. Arch. Otolaryng., 13:570 (April), 1931.

61. Stinson, W. D.: Osseous Atresia of the Posterior Choanae. Arch. Otolaryng., 15:101 (Jan.), 1932.

62. Srebrny: Cited by K. Vogel: Ztschr. f. Hals-Nosen- u. Ohrenh., 11:121, 1925.

63. Thacker-Neville, W. S.: Congenital Osseous Occlusion of Choanae. J. Laryng. & Otol., 42:48 (Jan.), 1927.

64. Thomasson: Congenital Bony Occlusion of the Right Nasal Choana. Laryng., 25:221 (April), 1915.

65. Tieman, H.: Ueber die Bildung der primitiven Choanen bei Saugethieren. Wurzburg, 1896.

66. Uffenorde, W.: Win Fall von Choanalatresie mit Demonstration. Ztschr. f. Laryng. Rhin., 1:475, 1908.

67. Vogel, K.: Ztschr. f. Hals-Nasen- u. Ohrenh., 11:121, 1925.

68. Von Schröetter: Ueber angeborenen kochernen Verschluss der Choanen. Monatsch. f. Ohrenh., Berlin, 19:97, 1885.

69. White, L. E.: Laryngoscope, 28:571, 1918.
Boston M. & S. J., 180:153, 1919.

70. Winslow, J. R.: Maryland M. J., 58:295, 1915.

71. Bailey, F. W.: Occlusion of the Nasopharynx. J. Iowa Med. Soc., 7:57 (Feb.), 1917.

72. Ballenger, W. L.: Malformation of the Pharynx. Stenosis of the Pharynx. Diseases of Nose, Throat and Ear. Lea & Febiger, 1925.

73. Curtis, B. F.: Cicatricial Structure of Pharynx: Cured by Plastic Operation. Annals Surg., p. 152 (Feb.), 1901.

74. Figi, F. A.: Stenosis of the Nasopharynx. Trans. Am. Laryng., Rhino. & Otol. Soc., 1929.

75. Goodyear, ? : Nasopharyngeal Atresia. Arch. Otolaryng., p. 974 (June), 1939.

76. Hall, F. J.: Adhesion of Soft Palate to Nasopharynx. British Med. J., 1:18, 1909.

77. Leland, G. A.: Cicatricial Structure of the Pharyngeal Orifice Relieved by Plastic Operation. Boston Med. J., 151:295 (Sept.), 1904.

78. MacKenty, J. E.: Nasopharyngeal Atresia. Arch. Otolaryng., 6:1 (July), 1927.

79. McIndoe, A. H.: Applications of Cavity Grafting. Surgery, p. 535 (April), 1937.

80. Nichols, J. E. H.: Sequelae of Syphilis in the Pharynx and Their Treatment. Tr. Am. Laryng. Assoc., 18:161, 1896.

81. Wright and Smith: Diseases of the Nose and Throat. Lea & Febiger, Phila., 1914.

LXXIV

AN ANATOMICAL STUDY OF THE NASOPHARYNX AS A PATHWAY TO THE INFRA-CAROTID PORTION OF THE PETROUS PYRAMID*

B. Russell Burke, M.D.

Atlanta

The fact that petrositis is receiving such a voluminous amount of discussion is an indication that this subject still occupies the center of the stage in otology and must be recognized as a clinical entity.

Pathology in the petrous bone has been recognized for many years. Teed[15] in a review of the history of petrositis reveals that the signs and symptoms were well recognized and methods of treatment were devised by 1904. What was probably the earliest study of petrositis was made by Edward Coch in 1838. In his article entitled "Contribution to the Pathology of Congenital Deafness," he reported pathological studies of the petrous pyramid.

The subject of this paper originated from a case in which an autopsy revealed an opening into the nasopharynx and also from an article by Guild[4] discussing "The Normal and Pathological Anatomy of the Petrous Pyramid" in a symposium before the American Otological Society. Guild was one of the first authors to mention that the nasopharynx should be taken into consideration in the diagnosis of lesions anterior to the labyrinth. He states that "inferiorly the petrous pyramid has certain relations of possible clinical importance. In the first place, it is very near to the lateral part of the vault of the nasopharynx. The relationship makes it reasonable to suppose that inflammatory reactions in this part of the pyramid might produce local reactions in the vault of the pharynx. So far as I know, the nasopharynx is not examined from this viewpoint in patients with infections of the petrous pyramid. Secondly, the tensor veli palatine and the levator palatine muscles do originate in part from the undersurface of the pyramid near the tip. So far as I am aware, no one has noted symptoms produced by irritations of these muscles."

---

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, 1940.

A few months after this article was published, it was my privilege to have a patient present a typical picture of petrositis a few weeks after I had done a complete simple mastoidectomy.

CASE REPORT*

Mr. R. V. W., aged 38, was first seen August 12, 1935, complaining of fullness in the ears. Both drums were inflamed but showed no signs of middle ear pressure or bulging. August 13, the left drum was bulging. Myringotomy was performed. On August 14, the right drum was bulging; myringotomy was also performed. The patient ran the usual course of otitis media—i.e., discharge, occasional pain in ears, and low-grade fever. Examination of pus revealed numerous Streptococci and Pneumococci. The right ear drained for about two weeks and gradually subsided, but the drainage in the left ear persisted with no fever, pain nor headache. Several roentgenograms were made showing the usual cloudiness and congestion of the mastoids. On September 19, 1935, a roentgenogram revealed a breaking down of the left mastoid, and on September 20, a complete simple mastoidectomy was performed. A leucocyte count at this time was 21,500; temperature was 102.6 degrees. The mastoid was an unusually large pneumatic type, and all the cells were broken down. There was an exposure of the dura of the middle fossa but no exposure of the lateral sinus. The postoperative period was characterized by considerable headache and pain around the left eye and temporal region of the same side (left). These symptoms subsided, and the wound healed nicely except that there was some drainage from the middle ear. On October 25 there was a severe headache (of deep-seated neuralgic type), and the patient noticed that he had double vision. A sixth nerve paralysis had developed. The patient complained of sore throat high up in the nasopharynx. A roentgenogram (see Fig. 1) revealed an extensive inflammation of the petrous pyramid, particularly at the apex and along the superior petrosal ridge.

The report of operation by the consultant was as follows: "At operation (typical unlocking type of operation), a small amount of pus in the angle of the petrosal and a few drops of pus in the labyrinthal cells were found, but there was a large osteomyelitic abscess at the apex in the dura, filled with pus and granulations, with a thickening of the dura which looked like a white exudate. The common carotid was ligated with fascia lata. The abscess showed on microscopic examination to contain a Streptococcus mucosus (Pneumococcic Type No. 3). The temperature rose to 106 degrees and came down, but he had a chill following transfusion. A lumbar puncture was done, and it was found that he had a rapidly spreading meningitis. Two days later his dura was opened because he developed severe pressure symptoms, and that night the patient died of meningitis." The autopsy report was: "Osteomyelitis of the whole petrous apex of the outer third of the sphenoidal base with a hole that went down into the throat. The eroding of bone involved the whole of the inferior and posterior surfaces of the apex, running from the medial fossa into the posterior fossa, as a serpiginous osteomyelitis, typical of a mucosal infection. In the center of the osteomyelitic area in the sphenoidal base, there was a hole that ran into the lateral wall of the nasopharynx from which drainage down was going on. This area was about the size of a quarter."

*Case reported by consultant.

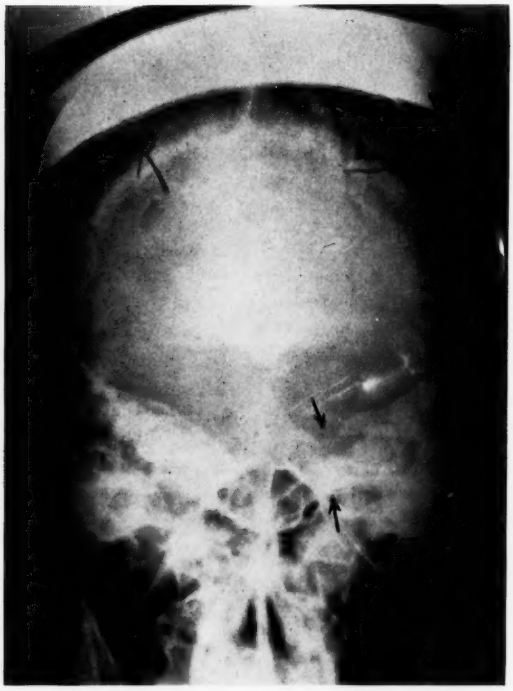


Fig. 1. This radiograph of the petrosal area shows normal aeration of the right mastoid, whose cells extend above the canal and nearly to tip of the petrous process. The left mastoid has been operated upon. A few cells are seen extending into the petrosal tip, nearly obscured by inflammatory reaction. There is also apparent erosion of the superior border of the petrosal tip on the left, bone destruction being demonstrable.

Conclusions: Necrosis in the left petrous tip.

This case presents several interesting features, foremost of which are the sore throat and pain in the nasopharynx. Although these symptoms did not appear of any particular significance at that time, we now, of course, realize their extreme importance.

Shortly after this I saw two other cases. One gave a history of a sudden rupture with the liberation of purulent material from the nasopharynx. This patient had had a complete simple mastoidectomy and three or four weeks afterward developed a sixth nerve paralysis. Roentgenograms were positive for a petrosal involvement. There is no definite way of proving this case because there was no injection of lipiodol into his nasopharynx to determine whether there was a fistulous tract, but I believe now that there was a spontaneous cure by rupture into the nasopharynx.

A third case was seen in our university clinic. A negro male was observed for two weeks with an acute otitis media and sclerotic type of mastoid. The acute symptoms subsided and no operation was performed, but drainage from the middle ear persisted for several weeks and suddenly ceased. The patient became very ill and was re-admitted to the hospital. No symptoms of meningitis had appeared. About one week after admission the patient developed a retropharyngeal abscess which was drained, and about one week later he was dismissed as well.

Although it is impossible to prove now that these last two cases come under the nasopharyngeal syndrome in which we are interested, it is believed that they belong to this group and were not recognized. It is for this reason that I believe many such cases have occurred and the true significance was not realized.

Sears[13] in his article "Otogenic Paralysis of the Abducens" was one of the first authors to correlate rather scattered data of this syndrome and was responsible for the rapid advance in diagnosis of petrositis. It is rather interesting to note in this article, "that of fifty-eight authors that gave a pathological diagnosis, nineteen attributed the infection of the sixth nerve to a circumscribed pachymeningitis at the apex of the petrous; nine to a serous or protective meningitis; four to inflammatory edema at the apex of the petrous; seven to edema of the brain with increased intracranial pressure; sixteen to toxic neuritis; four to extension of inflammation in the lateral sinuses and bulb by way of inferior petrosal sinus to region of the sixth nerve; one to isolated thrombosis of the superior petrosal sinus; two to extra-dural abscess; one to encephalitis; four to abscess at apex of petrous; one to hemorrhage into brain substance."

This article shows the extreme variations in opinion and diagnosis of this condition. The same is true now concerning the nasopharyngeal symptoms, which are being overlooked entirely or misinterpreted.

Infection of the inferior surface of the petrous pyramid is far from infrequent, as shown by Eagleton.[1] He states that "out of about fifty verified cases of suppuration within the petrous apex I have had six cases of osteomyelitis below the carotid. Of the six, only three caused a demonstrable fluctuating swelling in the pharynx. The other three cases presented large collections of pus below the carotid artery inaccessible to any operative approach from the aural region but probably curable by boldly perforating the bone of the petrous apex in the lateral pharynx roof, mesial and slightly posterior to the eustachian tube." (A more accurate method of locating the petrous tip, infra-carotid portion, will be demonstrated later.) A recent personal communication with Eagleton reveals that he has had eight cases rupture into the nasopharynx, two more than he has reported in his article.

Moorhead and Baker[8] reported a case of radical mastoidectomy on an acute exacerbation of a chronic ear. A peritonsillar abscess developed several days later. The abscess drained, and the patient was out of bed in about one week. About three weeks later the patient had a severe chill and symptoms of fulminating meningitis developed. At operation the medial fossa was exposed, pus was found, and a large hole in the cortex of the anterior side of the petrous pyramid was discovered. This case illustrates formation of an abscess outside and below the pyramid which burrowed itself into pharynx.

The authors state that this case shows the folly of draining an abscess from the inferior surface of the pyramid, but it is my opinion that if the significance of the abscess had been realized and there had been a prompt opening made with thorough investigation of the nasopharynx, and if nature's effort to drain this area had been assisted by surgical drainage through the nasopharynx, the patient would probably not have developed meningeal symptoms and an extensive operation been necessary.

Kopetzky and Almour in a personal communication stated that among fifty-nine cases of petrosal involvement they had four which ruptured through the pharynx. Three cases were reported, and one case is unreported. This case cured itself spontaneously without requiring operation.

A pioneer in the technique of exploring the petrous pyramid, Eagleton,[2] describes his unlocking of the petrous pyramid for localizing bulbar meningitis secondary to suppuration of the petrous apex. This is an extensive procedure, and sufficient elevation of the dura of the temporal sphenoidal lobe to reach the apex properly is made possible by extensive removal of the squama. His critics do not believe this approach is practical because exposure of the apical carotid portion of the petrous pyramid from this route cannot be accomplished in the living subject without lacerating the dura. However the mortality rate of most of the operators, regardless of their procedure, is about the same.

Kopetzky[5] describes an operative procedure by Almour. A tympano-mastoidectomy having been performed and a search for fistulas proven futile, a procedure was followed which consisted of removing the tympano muscle from its attachment and freeing the space between the eustachian tube and the channel of the tensor tympani and entering the petrosal pyramid at this point. This is accomplished by use of an electric burr of 1.5 mms. placed high up at a level with the roof of the musculo-tubal canal, one-sixteenth of an inch posterior to the mouth of the eustachian tube and directed at an angle of twenty-two degrees to the external auditory canal. This attack is directed into the body or base of the pyramid, and while it does drain some of the apex cells, I doubt if any of the infracarotid portion of the petrous apex is reached. There is also danger of injury to the carotid artery and cochlea.

Ramadier[11] schematically distinguishes three localizations of osteitis in the petrous pyramid—posterior petrositis, anterior petrositis, and apicitis—and described an operative technique for each. It is with the latter that we are concerned. Ramadier describes this as a paracarotid approach. This is done by resecting the anterior and inferior walls of the bony auditory canal. This resection can be done without damage to the temporo-mandibular articulation, because there is between it and the temporal bone space which is filled with cellular fibrous tissue.

Critics of this technique declare that it defeats its own purpose because drainage is accomplished by an opening in the internal wall of the carotid canal, and this becomes automatically obstructed when the carotid artery is allowed to fall back in its canal.

Guild's[3] criticism of Ramadier's operation and also Lempert's procedure is that adhesions of the carotid artery to the wall of the osseous canal occur; especially is this true, he points out, when the

adherent area is calcified or excessively thin, a condition which constitutes an operative hazard that has hitherto been overlooked.

Lempert[6] describes an operation for apicectomy and also four stages of this procedure:

1. The performance of a mastoido-tympanectomy.

2. The exposure and inspection of the basal labyrinthine portion of the petrous pyramid.

3. The creation of a surgical intra-petrosal approach to the apical carotid portion of the petrous pyramid.

4. The complete exenteration of the bony cellular structure of the apical carotid portion of the petrous pyramid accomplished through the exploration of the entire apical course of the internal carotid artery.

A review of this technique reveals that this procedure is a one-stage operation for anterior, posterior and apical lesions of petrositis. Radical as it may seem, I wonder after all if it isn't more rational and certainly more complete than any procedure so far described. However I am sorry to say that there are few operators that can carry out this technique.

Myerson, Blumberg and Rubin[9] have devised a method of approach for osteomyelitis of the inferior aspect of the petrous pyramid by way of a lateral cervical region through the retropharyngeal space.

This method of approach, to my mind, is a rather indirect route because there is an exposure of the carotid sheath and its contents and many large vessels and muscles to resect. Why do this when there is a more simple and direct route?

DIAGNOSIS

As to the diagnosis of apicitis, per se, this would be quite a clinical problem because it is difficult in some cases to determine whether the infection has extended into the pars petrosa itself or not.

Mayer[7] states that of all the symptoms headaches are most constant. These headaches are very characteristic, radiating mostly to the temporal region and anteriorly to the eye of the diseased side. These pains are extremely intensive and are pronounced neuralgiform in character. They show evidence of an inflammatory process in the apex of the petrous bone near Gasser's ganglion.

Schugt[12] reports a case of glossopharyngeal neuralgia associated with abscess of the petrous tip following mastoiditis. Vernet's syndrome or the syndrome of the jugular foramen is discussed in this article. The syndrome is characterized by difficulty in swallowing, anesthesia or hyperesthesia of the soft palate and the pharynx, loss of taste in the posterior part of the tongue (glossopharyngeal nerve), paralysis of the vocal cord on the affected side (vagus nerve), or disturbance of the function of the sternocleidomastoid and trapezius muscles (spinal accessory). The inflammatory process in the antelabyrinth may involve one or all three of the nerves. The glossopharyngeal, lying close to the bony structure of the antelabyrinth and separated from the others by dura mater, is most likely to be affected before the other nerves. I think this syndrome is one of the most important diagnostic signs to be brought to our attention.

Ramadier[40] differentiates the diagnosis of anterior and posterior lesions of petrositis according to location of drainage following, of course, a mastoidectomy. The posterior petrosites are recognized by the drainage from the mastoid wound, while the canal is dry and the tympanum tends to acquire its normal appearance. The anterior petrosites are manifested by a tympanal suppuration while healing of the mastoidectomy takes place normally. These signs are accepted generally as being of definite diagnostic value.

Temperature is an inconstant factor; usually it is of a low-grade septic type. Normal temperature for two or three successive days is not unusual in the less severe types.

Paralysis of the sixth nerve (abducens) as we know does not always take place in petrous pyramid suppuration, but when it does and is associated with other symptoms we should certainly consider it as of definite diagnostic value and as rather indicative of the infection involving the tip or infra-carotid portion of the petrous pyramid. This paralysis may be brought on by swelling and congestion of the dura over Dorello's canal, and when symptoms of recession take place, the paralysis clears up.

Taylor[14] states that the popular positions for demonstrating suppurative lesions in the antelabyrinth are axial projections of the base of the skull and oblique views. With the axial projections the pyramids are x-rayed simultaneously and, I believe, have an advantage over the oblique views (Stenver) which are x-rayed separately.

Stereoscopic views should be made when possible, and there should also be a recording of the exposure factors in each case so

that the same technique may be used in re-examinations. At present regional or sectional radiography by the laminagraph is being developed. Particularly will this be useful in diagnosis of suppurations in the antelabyrinth.

INDICATIONS FOR OPERATION IN NASOPHARYNX

The most important diagnostic signs of antelabyrinthine suppurations have been mentioned. When all of these have been taken into consideration and the symptoms of progression of the disease are becoming more manifest, then certain surgical procedures should take place. This is true, of course, only after a thorough revision of the mastoid wound and a search for fistulas have been made and, if necessary, a tympano-mastoidectomy and a further search for fistulas. In this connection I do not understand the objections to the sacrifice of hearing when a human life is at stake. The best method is to decide in one's own mind which is more important and which one would choose if he were the patient. A nasopharyngeal approach should take place after the above procedures have been followed.

Nasopharyngeal examinations should be made at regular intervals, particularly if the symptoms indicate that we are dealing with an anterior petrositis. These examinations should be made with a naso-pharyngoscope or better still by digital examination with a gloved forefinger. I believe that with the digital examinations one can determine better the amount of swelling, if any, over the longus capitis muscle at its attachment and certainly determine the degree of pain or tenderness on pressure over this muscle, just as one elicits tenderness in the mastoid by pressure over either the mastoid antrum or tip.

THE NASOPHARYNGEAL OPERATION

The objective of this procedure is the plate of bone which forms the floor of the carotid canal.

Anesthesia may be local or general but preferably general because a mouth gag will have to be used and consequently the patient will have to be fairly well relaxed. It would be better to use a mouth gag with a self-retaining tongue depressor so that the assistant may be able to use one hand for suction and assist with the other.

An incision is made through the soft palate to the right or left of the uvula, depending upon the side to which the operation is directed (Fig. 2). The incision is carried vertically all the way

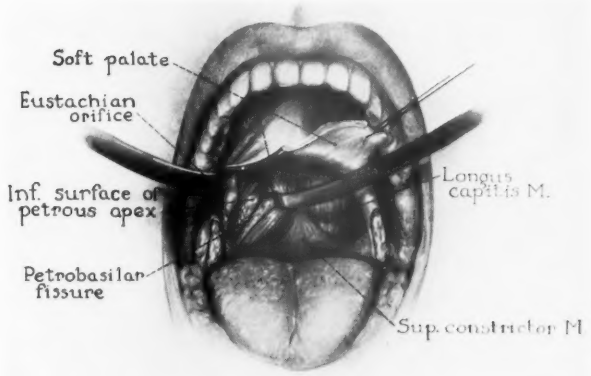


Fig. 2. Showing operative procedure through nasopharynx. All drawings with the exception of Fig. 3 are made from original dissections by the author.

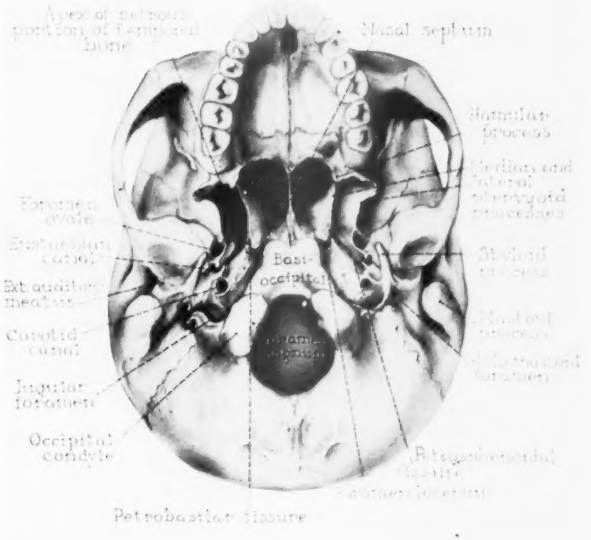


Fig. 3. Inferior surface of skull showing relationship of inferior aspect of petrous pyramid. This view of skull demonstrates relationship of median pterygoid process to infra-carotid portion of petrous apex, and the relative angles of the internal carotid canal with the basi-occipital bone.

through the soft palate. Black silk sutures are placed on each side and the soft palate retracted in this manner. The whole of the naso-pharynx is then exposed. With the forefinger the medial pterygoid process is palpated, and this process is followed downward to the basi-occipital bone which can be easily felt, and if the finger is moved slightly forward, the petro-basilar fissure can be distinguished. Just laterally the inferior aspect of the petrous bone is easily distinguished. A vertical incision of about two cms. is made at this point.

The structures through which the incision is made are: (1) mucous membrane; (2) pharyngeal aponeurosis; (3) the longus capitus muscle itself (see Fig. 2). The fibers of this muscle are quite dense because it is at the attachment of the basi-occipital bone and the medial side of the inferior aspect of the petrous bone that these fibers form its tendon. The fibers of the levator veli palatini muscle are also resected laterally, and the infra-carotid portion of the petrous bone is plainly brought into view. The tendinous sheath of the levator veli palatini is attached to the lateral margin of the inferior petrosal surface, and the carotid canal lies just posterior (see Fig 4). These muscle attachments are stripped partly away with an elevator, and there is an exposure of bone of about 1 to 2 cms. between the carotid canal and the foramen lacerum which is filled with tough fibro-cartilage. No large blood vessels are encountered in this operative field. There are only very small branches from the ascending pharyngeal artery and also very small branches from the palatine artery, which is a branch of the external carotid.

The objective of this procedure is this plate of bone on the inferior aspect of the petrous tip, and if disease were present either pus or softened bone would be encountered, and adequate drainage could be easily secured.

In this anatomical study, twenty-two adult cadaver heads were dissected, this procedure being carried out on both sides of each head. It was interesting to note that on gross examination only four specimens showed any signs of pneumatization on the inferior aspect of the tip. No sections were taken for microscopic examination because it was not the intention of the writer to discuss this phase. A competent anatomist examined these specimens, and he was of the same opinion that pneumatization had taken place in only four. This seems to confirm Eagleton's opinion that the inferior aspect is composed of cancellous or compact bone containing marrow and that the lesion being dealt with is an osteomyelitis.

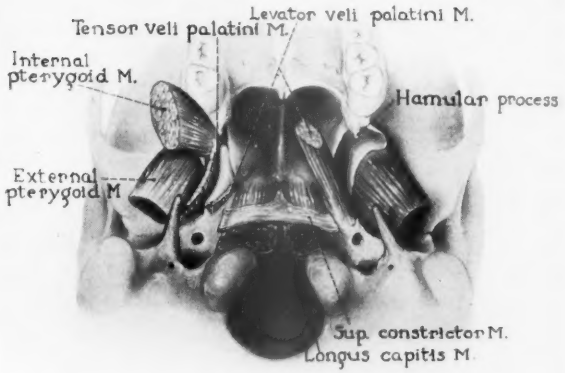


Fig. 4. Showing the origin and insertion of important muscles in the nasopharynx.

Superior Constrictor Muscle: Origin—Palate bone, internal pterygoid plate, pterygomaxillary ligament, alveolar process of jaw and side of tongue. Insertion—Posterior median raphe.

Internal Pterygoid Muscle: Origin—Pterygoid fossa of sphenoid bone, tuberosity of maxilla. Insertion—Inner surface of ramus and angle of mandible.

External Pterygoid Muscle: Origin—External pterygoid plate, great wing of sphenoid. Insertion—Condyle of mandible.

Levator Veli Palatini Muscle: Origin—Apex of petrous portion of temporal bone and cartilaginous eustachian tube. Insertion—Aponeurosis of soft palate.

Tensor Veli Muscle: Origin—Scaphoid fossa, spine of sphenoid, vaginal process of temporal. Insertion—Inner surface of ramus and angle of mandible.

Longus Capitis Muscle: Origin—Anterior tubercles of transverse processes of the 3rd, 4th, 5th and 6th cervical vertebrae. Insertion—Basilar process of occipital bone.

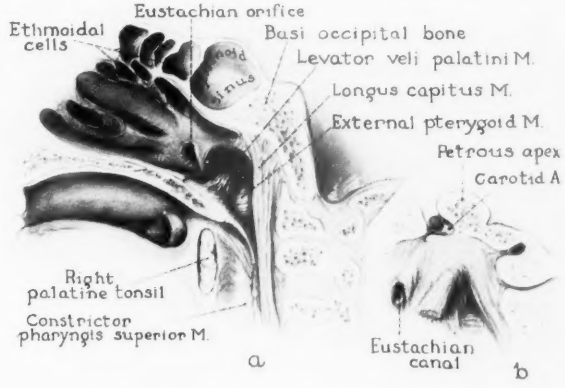


Fig. 5. Sagittal section of one of the dissected specimens showing relation of carotid artery and attachment of longus capitus muscle to basilar process of the occipital bone, and the attachment of the levator veli palatini muscle to the infra-carotid portion of the petrous apex.

If softened bone were encountered this could be easily removed with a curet. I do not think that it would be advisable to attempt to remove the internal carotid artery from its canal in this procedure, first, because sufficient drainage could be accomplished by curettement of this osteomyelitic area, and secondly, because the operative field is too small to attempt to manipulate the artery in the presence of this diseased bone.

In this anatomical study important measurements relative to the approach to the inferior aspect were taken. This included the distance from the base of the pterygoid hamulus to the foramen lacerum, which is of utmost importance in determining the site of the incision, and the angle of the internal carotid canal with a vertical line drawn through the center of the basi-occipital bone. This angle is important because it gives an idea as to the location of the carotid canal.

Forty-four adult skulls were studied in this respect. Bilateral measurements were taken.

It is interesting to note that in the distance from the base of the pterygoid hamulus to the foramen lacerum, the longest measurement was 4.1 cm., and the shortest was 2.7 cms., a difference of only 1.4 cm. Of the bilateral measurements the greatest difference was only .8 cm.

There was also a marked uniformity in the angle of the internal carotid canal with a vertical line drawn through the center of the basi-occipital bone. The greatest difference in angles between the two sides was only 5 degrees. Of the whole group the greatest differences were in a skull which measured a 40 degree angle on the right side, another which measured a 58 degree angle on the left side —a difference of only 18 degrees in the whole group.

This presentation has been an anatomical study of the nasopharynx, based on dissections of 22 adult cadaver heads and measurements of important angles of 44 adult skulls.

Further studies should be continued in order to improve our knowledge and techniques in treatment of these very difficult conditions.

SUMMARY

1. Petrous pyramid suppuration is a clinical entity and must be recognized as early as possible.

2. There are many cases of abscess formation in the nasopharynx associated with suppuration in the antelabyrinth, the significance of which has not been realized.

3. Especial attention must be directed to the nasopharynx in these cases, with examinations of the nasopharynx by either a nasopharyngoscope or by digital examination at regular intervals.

4. There is a simple and direct route by which infections in the infra-carotid area may be drained.

While this is a preliminary anatomical report, I hope this procedure will be justified by future clinical results.

478 PEACHTREE STREET.

BIBLIOGRAPHY

1. Eagleton, Wells P.: Osteomyelitis of the Inferior Surface of the Petrous Pyramid. J. A. M. A., 107:482, 1936.

2. Eagleton, Wells P.: Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex. Arch. Otolaryng., 13:386, 1931.

3. Guild, Stacy R.: Hitherto Unrecognized Danger in the Operation of Ramadier for Suppuration of the Petrous Pyramid. Acta oto-laryng., 25:561, 1937.

4. Guild, Stacy R.: II. Normal and Pathological Anatomy of the Petrous Pyramid. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 44:1011, 1935.

5. Kopetzky, Samuel J.: The Surgical Treatment of Various Types of Lesions in the Petrosal Pyramid. Surg., Gynec. & Obst., 66:395, 1938.

6. Lempert, Julius: Complete Apicectomy (Mastoidotympano-apicectomy). Arch. Otolaryng., 25:144, 1937.

7. Mayer, Otto: Fortschritte auf dem Gebiete der operativen Ohrenheilkunde (Freilegung der Arteria carotis interna). Wien. klin. Wchnschr., 51:996, 1938.

8. Moorhead, Robert L., and Baker, John P.: Suppuration of the Petrous Pyramid: When and How to Operate. Report of Thirty Cases. Arch. Otolaryng., 28:497, 1938.

9. Myerson, Mervin C., Blumberg, Ralph, and Rubin, H. W.: A Proposed Operation for Osteomyelitis of the Inferior Aspect of the Petrous Pyramid. Arch. Otolaryng., 25:373, 1937.

10. Ramadier, J.: Les indications operatoires dans les petrosites. Oto-rhino-laryng. internat., 21:489, 1937.

11. Ramadier, J.: A propos du traitement des petrosites. Ztschr. f. Hals-, Nasen- u. Ohrenh., 40:524, 1937.

12. Schugt, Henry P.: Glossopharyngeal Neuralgia Associated with Abscess of the Petrous Tip Following Mastoiditis. Arch. Otolaryng., 26:321, 1937.

13. Sears, William H.: Otogenic Paralysis of the Abducens, with Especial Mention of Isolated Palsy Associated with Irritation of the Gasserian Ganglion. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 35:348, 1926.

14. Taylor, Henry K.: Suppuration in the Pneumatic Antelabyrinthine Portion of the Petrous Pyramid. New York State J. Med., 37:231, 1937.

15. Teed, R. Wallace: A Historical Note Regarding Petrositis. Arch. Otolaryng., 23:692, 1936.

LXXV

THE TREATMENT OF CHRONIC OBSTRUCTIVE RHINITIS WITH SUBMUCOSAL INJECTIONS OF SODIUM PSYLLIATE*

E. A. Thacker, M.D.

New Orleans

Intermittent and constant chronic nasal obstruction is a common complaint for which patients seek the advice of the oto-rhino-laryngologist. Associated with this complaint may be postnasal dripping, headache, and pain in the frontal, temporo-parietal regions, and occasionally in the postauricular area. It is not unusual for one or more of these symptoms to be so sufficiently pronounced as to overshadow the nasal obstruction.

Chronic nasal blockage may be the result of anatomic abnormalities of the septum, bony framework of the turbinates, or such tumors as adenoidal hypertrophy, polyps, papillomata, fibromata, or malignancies. The correction of such conditions requires appropriate surgical, radium or x-ray treatment and does not fall into the scope of this manuscript.

This investigation has been confined to conditions of the nasal mucosa which are responsible for chronic nasal obstruction and its associated symptoms. There has been an overlapping in the nomenclature because these conditions are classified according to the etiological, pathological and clinical findings. To eliminate this confusion, a simple classification is used here into which all of the common nonspecific obstructing forms of rhinitis can be grouped. At least it serves the purpose of deciding which patients are subjects for the treatment described hereinafter. This classification includes:

1. Chronic obstructive rhinitis which shrinks well with astringents.

2. Definite allergic rhinitis.

3. Chronic obstructive rhinitis which does not shrink well with astringents.

---

*From the Department of Graduate Studies, Tulane University Medical School and the Eye, Ear, Nose and Throat Hospital.

The obstructive type of rhinitis which shrinks well and belongs to the non-allergic and non-specific group is the most satisfactory for treatment. Those patients who are definitely in the allergic group should be treated by desensitization and the removal of the offending activator if possible. Should this fail, this form of therapy might prove to be beneficial. The chronic rhinitis which fails to shrink well with cocaine, adrenalin or ephedrine is not amenable to treatment of sclerosing agents, administered by submucosal injections into the turbinates, such as phenol,[1] alcohol,[2, 3, 4, 5] glycerine, quinine and urea hydrochloride,[6, 7] dextrose[6, 8] and sodium morrhuate.

A review of the literature leaves one confused as to the effect of most of these preparations and methods of treatment. Fatty acid derivatives have been found superior to the other sclerosing agents in the treatment of varicosities, hydroceles, and hernias. Fishof[9] reported seven cases of rhinitis which he successfully treated by the submucosal injection of sodium morrhuate. A series of 58 cases of chronic simple, hypertrophic and vasomotor rhinitis treated with this preparation has been reported recently by me.[10] Another mild soap of a fatty acid, sodium psylliate, has been used in the last few years by investigators for the treatment of various conditions. Biegeleisen,[11] Biederman,[12] McCastor and McCastor,[13] Rice,[14] Rice and Mattson,[15] and Riddle[16] have enthusiastically endorsed it for the treatment of hernia. Schultz[17] reported excellent results with this preparation in overcoming subluxation of the joints. Favorable results were obtained by Biederman[18] on cases of hydrocele, and Biegeleisen[19] and Watson[20] in obliterating varicose veins. Rice, Rice and Mattson, Schultz, and Biegeleisen present animal and clinical evidence that this substance has a low toxicity and a wide range of safety. Histologic studies by Rice, Schultz, McMillan and Cunningham[21] and Manoil[22] reveal that an early lymphocytic infiltration occurs which is replaced later by fibrous tissue. No sloughing or other undesirable reactions were noted when sodium psylliate was injected into the muscle, subcutaneous tissue, joints or veins. There have been no reports of allergic sensitivity to this drug during the five years it has been available for use.

The good results obtained in the treatment of other conditions with sodium psylliate have prompted this investigation with the mild soap in certain cases of chronic rhinitis. Sodium psylliate is a five per cent solution of the sodium salt of a vegetable oil extracted from psyllium seed.

Before considering a patient suitable for this treatment, it should be emphasized that a thorough history must be taken with particular

attention being given to sinus disease and allergy. A physical examination of the nose should be made, including transillumination of the sinuses and x-ray if indicated. Anatomical defects of the nose and sinus disease should receive appropriate treatment. Ocular disorders as a source of headache and associated symptoms should be ruled out. One should always bear in mind that other bodily disorders may cause headache and symptoms referable to the head.

The indications for treatment with sodium psylliate are the same as for sodium morrhuate. These are:

1. Chronic nasal obstruction from engorged turbinates which have failed to respond to the usual therapeutic measure after one month of trial.

2. Postnasal dripping, chronic pharyngitis, headache and neuralgic pains in the head and neck in which an intumescence of the turbinates exists and is causing pressure on the lateral wall of the nose or the septum.

3. Intermittent nasal obstruction of long duration, especially when this is associated with temperature and humidity changes.

4. Long standing cases of chronic sinusitis with associated turgescence of the turbinates.

5. The turbinates must shrink well with astringents.

This form of treatment is contra-indicated in the following conditions:

1. Chronic systemic diseases such as hypertensive or other types of advanced cardiac disease, arteriosclerosis, nephritis, and such specific diseases and allergy in which the proper treatment of the primary cause should correct the nasal condition.

2. Anatomical defects of the nasal passages. Such abnormalities should be corrected surgically; then if the nasal obstruction persists after an interval of a few months, the engorged turbinates should be considered for submucosal injection of the sclerosing agent.

3. Since there is little or no shrinkage of the soft tissues in true hyperplastic rhinitis, such patients will not respond to this type of therapy.

The technic used for the submucosal injections of sodium psylliate is essentially the same as for the sodium morrhuate.

1. A two or four per cent butyn solution on two tampons is placed around the turbinate to be injected.

2. A No. 22 spinal puncture needle on a tuberculin syringe is very satisfactory for injecting the inferior turbinate. A long gold needle which can be properly bent to fit the individual case is advantageous for injecting the middle turbinate.

3. One-fourth to one-half cubic centimeter of sodium psylliate is drawn into the syringe. This can be varied according to the individual case.

4. The tampons are removed. The bevel of the needle is turned away from the bony framework of the turbinate. The anterior portion of the mucous membrane is pierced, and the needle is inserted toward the posterior end of the turbinate. The sclerosing substance is liberated as the needle is gradually withdrawn.

5. A pledget of cotton is placed in the nose in order to stop the bleeding from the needle puncture.

6. The cotton is removed in five minutes and a tampon saturated with neosynephrin or ephedrine is placed around the turbinate for ten minutes.

7. The patient is advised to use a one per cent ephedrine or neosynephrin solution every two hours for five days.

8. Only one side of the nose is injected at a sitting. After one week the other side may be treated.

9. Two or three weeks should elapse before another injection is made into the same tissue.

Butyn is used because it has no vasoconstricting action. A two or four per cent solution applied by tampon produces a good anesthesia. No toxic effects have been noted with this preparation. A ten per cent larocaine solution can be used as a topical anesthetic; even though slight shrinkage occurs this does not interfere greatly with the injection. It is much easier to inject the sclerosing agent if the turbinates are not shrunken. Furthermore, the oil is given a better chance to disseminate throughout the submucosal tissue. The soft tissues are shrunken with saturated tampons of ephedrine or neosynephrin about five minutes after the injection on the theory that this would aid in the distribution of the sclerosing substance into areas which otherwise would not be reached.

Occasionally some of the sodium psylliate may escape by accident into the nasopharynx. The patient states that a slight burning sensation occurs, but that no disagreeable taste is noticed. Very little pain or discomfort is experienced following the injection. An increase in the mucoid discharge occurs for about two days. By the third or fourth day a gradual improvement in breathing through the nostril begins.

RESULTS

The nasal obstruction was relieved in the twenty-four patients treated with sodium psylliate. The associated symptoms were either entirely relieved or improved. No destruction or ulceration of the nasal mucous membrane has occurred in any of the subjects treated. Neither have any adhesions been observed. The number of injections necessary to produce the desired effect ranged from one to four in the same turbinate, with an average of two injections being sufficient in the majority of instances.

The advantages of the use of sodium psylliate and sodium morrhuate over the other forms of nasal treatment for the chronic type of rhinitis which shrinks well to astringents are:

1. The technic of administration is simple and easy to master.

2. No expensive equipment is necessary. One five cc. rubber-capped vial of the sclerosing preparation is more than sufficient for the entire treatment of one patient.

3. No ulceration or adhesions have been noted following this treatment.

4. The physiological function of the nose is disturbed only during the first few days after treatment. This is controlled by vasoconstrictor medication. The mucous membrane tends to return to normal.

5. Very little pain or discomfort is experienced by the patient following the injection.

REPORT OF CASES

Since several of the patients presented similar symptoms, only a few of the more interesting case reports are submitted. In order to save space, only the positive clinical and laboratory finding are reported on these cases.

CASE 1.—R. M. This white woman, aged 50, complained of headache and nose blockage which had been present for almost 10

years. These symptoms were so aggravating that she was unable to sleep well. She also complained of a chronic sore throat and postnasal dripping. A submucous resection was done nine years ago. She had been treated constantly from 1931 to the present time with conservative measures, including trichloracetic acid applied to the inferior turbinates. Several x-ray films during this period showed some involvement of the ethmoids and antra. The antra were washed, and only a small amount of mucoid material was obtained. Physical examination revealed rather pale congested inferior turbinates which shrank well with astringents. A slight mucoid discharge was seen by posterior rhinoscopy. Chronic pharyngitis was present. On March 4, 1940, tampons of butyn were applied after which one-fourth cc. of sodium psylliate was injected into the left inferior turbinate. There was a good deal of blockage but very little pain or discomfort following the injection. Three days later, one-third cc. was inserted into the right inferior turbinate. The patient returned nine days later breathing much better. At this time, one-half cc. sodium psylliate was put into the left inferior turbinate. One week later there was a marked improvement in breathing. The right inferior turbinate was still engorged, so one-half cc. of the oil was injected into it. After two weeks this patient stated that she had an increase in the nasal discharge, but that she was feeling much better. There was still slight nasal blockage occasionally. A week later, one-half cc. was injected into the left inferior turbinate. On April 19th she volunteered the information that she was able to sleep now without being disturbed by headache and nasal obstruction. More than five months has elapsed since the completion of this treatment. The patient has no complaints. The mucous membrane appears healthy and normal.

Case 2.—A. D. This colored female, aged 45, complained of a frontal and occipital headache, frequent head colds, and postnasal dripping which had been present for five years. The symptoms were aggravated when her nose was stopped up. Glasses were worn without any relief. The left inferior turbinate was large and boggy, more so than the right. They both shrank well with astringents. Conservative treatment for three months failed to give this patient any permanent relief. On March 3, 1940, larocaine tampons were used and one-third cc. sodium psylliate was injected into the left turbinate. One week later, she stated that some pain in the teeth and left maxilla for three hours followed the last injection. Butyn tampons were applied and one-half cc. of the oil was inserted into the right inferior turbinate. On March 20th, she stated that the headaches had dis-

appeared and that she was breathing better. Butyn was applied and one-half cc. of the sclerosing agent was injected into the left turbinate because it was still enlarged and shrank well. This patient has been seen at intervals since then. Five months have passed without a recurrence of any of the symptoms, except that a slight postnasal dripping has continued.

CASE 4.—I. G. This colored male, aged 40, was referred from the eye department because of glaucoma and chronic conjunctivitis. He had frequent headaches, nose blockage at night and hawking and spitting. A chronic pharyngitis and tonsillitis were present. The right inferior turbinate was markedly turgescent and pale and completely occluded the nostril. X-ray of the sinuses showed some opacity of the right antrum. There was no seasonal variation in the nasal obstruction, although he usually had more watery discharge and occasional sneezing during the summer. Nasal smears revealed neutrophiles, small lymphocytes and an occasional eosinophile. On March 7th, larocaine tampons were applied and one-half cc. of sodium psylliate was injected into the right inferior turbinate. One week later, the left inferior turbinate was completely occluding the nostril. Butyn was used, and one-half cc. of the oil was injected into this turbinate. On March 25th, one-half cc. was inserted into the right turbinate. One week later, this patient was breathing much better through both nares. There was no blockage complained of at night, and only occasionally was there hawking and spitting. The left inferior turbinate was still somewhat turgescent, so one-half cc. was injected into it. The patient returned 18 days later, because of some engorgement, for one-half cc. into the right inferior turbinate. Because the patient was symptom-free, no further injections were made until June 27th, even though there was still turgescence present. On this date, one-half cc. was inserted into the right turbinate, and one week later the same amount was used for the left inferior turbinate. Two months have elapsed since the last injection was made. The patient is still free from all of his previous symptoms, except an occasional pain in and around the right eye which is attributed to the glaucoma. The turbinates at this time appear more normal than prior to treatment, although there apparently is a slight hyperplasia of the right inferior turbinate.

CASE 5.—M. S. This white woman, aged 24, sought advice because of headache, pain between the eyes, and a postnasal dripping. She had had nasal obstruction for one year, especially on the left side of the nose. The left middle turbinate had been crushed with slight benefit. She received treatment for six months without any perma-

nent benefit. An x-ray taken six months ago showed some post-ethmoidal involvement. Examination revealed a boggy, reddened left inferior turbinate which shrank well. On March 18th, after butyn anesthetic one-half cc. of sodium psylliate was injected into the left inferior turbinate. She was seen one week later and stated that only one headache had occurred during that week. There was less nasal obstruction. The patient has returned regularly for observation, and six months have now passed with no recurrence of the original symptoms. The mucous membrane of the left inferior turbinate appears normal.

CASE 13.—A. P. This colored female, aged 32, was under treatment in the clinic for 10 years by shrinkage methods, trichloracetic acid, alcohol, and Dowling tampons with no benefit. Her chief complaint was nasal obstruction for 12 years, severe headaches and pains in the frontal and postauricular regions, marked postnasal dripping with hawking and spitting all day long, and tinnitus. She stated that she was unable to sleep well at night because of these symptoms. An increase in the watery discharge from the nose but no sneezing was noticed during the summer. The patient is sensitive to shrimp, pineapple, oysters, fish, crabs and strawberries. An allergic dermatitis occurred in February, 1940. On several occasions, the headache and pain were relieved by cocainizing the sphenopalatine ganglion. Examination revealed large, boggy, rather pale inferior turbinates. A spur was present on the nasal septum, but the patient refused operation at this time. One June 29th, after butyn anesthetic, one-half cc. of sodium psylliate was injected into the right inferior turbinate. There was some discomfort in the right cheek for about two days following the injection. A similar injection was made into the left inferior turbinate one week later. The patient returned on July 13th, at which time she stated that no more headaches had occurred and that she was sleeping well all night without having to use "nose drops". The sneezing and watery discharge had ceased, and the tinnitus had disappeared. There was very little postnasal dripping. She has returned regularly for observation. Two months have passed since the last injection, and so far there has been no recurrence of symptoms. The mucous membrane is not nearly so pale as it previously was, and the turbinates have been reduced to practically normal size.

SUMMARY

1. Twenty-four patients with chronic obstructive rhinitis, which shrank well with astringents, have been treated successfully by the submucosal injection of sodium psylliate.

2. The associated symptoms of headache, sphenopalatine ganglion neuralgia, and postnasal dripping were either relieved or markedly improved.

3. It appears that long standing refractory cases of sinusitis in which there is a chronic engorgement of the turbinates are benefited by this form of therapy.

4. A reduction in the size of the turbinates as well as an improvement in the appearance of the mucous membrane results from the treatment with this preparation. No deleterious effects have followed this method of treatment.

5. The technic of administration and the method of choosing the patients who will respond to this form of therapy are described.

6. Further observation and investigation are to be continued in order to evaluate the relative merits of the two fatty acid esters, sodium psylliate and sodium morrhuate.

EYE, EAR, NOSE AND THROAT HOSPITAL.

BIBLIOGRAPHY

1. Hutchinson, C. A.: Sclerosing Submucosal Injections of Turbinates. Brit. M. J., 1:72 (Jan.), 1937.

2. Hutter: Heilung eines Falles von Rhinitis nervosa durch endonasale Alkoholinjektion. Monatschr. f. Ohrenh., 58:663, 1924.

3. Metzenbaum, M.: Alcohol Injections into Inferior Turbinates for Amelioriation of Symptoms of Common Cold, Rose Fever, Hay Fever, and Hay Fever Asthma. Laryngoscope, 44:182 (Mar.), 1934.

4. Leichsenring: Behandlung allergischer Krankheiten mit Alkoholinjektion in die Nasenschleimhaut. Ztschr. f. Hals-, Nasen- u. Ohrenh., 28:220 (May), 1931.

5. Vail, H. H.: Treatment of Hay Fever by Nasal Injections of Alcohol. Arch. Otolaryng., 18:651 (Nov.), 1933.

6. Ibanez, L. M. Garcia: El Tratamiento conservador En Las Rhinitis Hipertroficas. Bol. Asoc. med. de Puerto Rico., 25:544 (Sept.), 1933.

7. Monson, L. P.: Treatment of Hypertrophied Inferior Turbinate by Use of Sclerosing Solutions. Arch. Otolaryng., 22:96 (July), 1935.

8. Baer, M.: Zur Injektionsbehandlung der Rhinitis chronica hyperplastica. Monatschr. f. Ohrenh., 73:108 (Feb.), 1939.

9. Fishof, F. E.: The Treatment of Vasomotor Rhinitis and Allied Conditions with Sodium Morrhuate. Arch. Otolaryng., 27:413 (Apr.), 1938.

10. Thacker, E. A.: Sodium Morrhuate in the Treatment of the Chronic Simple, Vasomotor, and Hypertrophic Forms of Rhinitis. Accepted for publication in Arch. Otolaryng.

11. Biegeleisen, H. I.: Two Fatty Acid Solutions for the Injection Treatment of Hernia. Am. J. Surg., 37:413 (Sept.), 1937.

12. Biederman, M.: Pitfalls in Injection Treatment of Hernia and How to Avoid Them. Med. Rec., 146:253 (Sept.), 1937.

13. McCastor, J. T., and McCastor, Mary: Injection Method for the Treatment of Hernia: Preliminary Report. Virginia M. Monthly, 65:744 (Dec.), 1938.

14. Rice, C. O.: The Rationale of the Injection Treatment of Hernia. Minn. Med., 18:623 (Sept.), 1935.

15. Rice, C. O., and Mattson, H.: Histologic Changes in the Tissues of Man and Animals Following the Injection of Irritating Solutions Intended for the Cure of Hernia. Ill. Med. J., 70:271 (Sept.), 1936.

16. Riddle, P.: The Injection Treatment of Hernia. Texas State J. Med., 32:226 (July), 1936.

17. Schultz, L. W.: A Treatment for Subluxation of the Temporomandibular Joint. J. A. M. A., 109:1032 (Sept.), 1937.

18. Biederman, M.: Treatment of Hydrocele with Sylnasol. Med. Rec., 147:80 (Jan.), 1938.

19. Biegeleisen, H. I.: Fatty Acid Solutions for the Injection Treatment of Varicose Veins. Ann. Surg., 105:610 (Apr.), 1937.

20. Watson, G. B.: Effect of Pregnancy on Varicosities of the Lower Extremities Proc. Sixth Post-Coll. Assembly, Ohio State U., p. 247 (Mar.), 1939.

21. McMillan, W. M., and Cunningham, D. R.: The Injection Treatment of Reducible Hernia. J. A. M. A., 106:1791 (May), 1936.

22. Manoil, L.: Histologic Effect of Various Sclerosing Solutions Used in Injection Treatment of Hernia. Arch. Surg., 36:171 (Feb.), 1938.

LXXVI

OSTEOMA OF THE NOSE AND ACCESSORY SINUSES WITH REPORT OF CASES*

W. Likely Simpson, M.D.

AND

Russell I. Williams, M.D.

Memphis

Osteomas are in a true sense benign, slow growing, usually encapsulated bony new growths which have a tendency to displace adjacent structures. They are usually attached to the bone from which they are derived. Malignant tendencies may be manifested because there is no limitation in the amount of displacement or erosion of surrounding tissues that this bony neoplasm may produce.

The number of osteomas of the nasal accessory sinuses and nasal cavities as reported in literature attains considerable figures, but surprisingly little is mentioned in regard to the pathogenesis of this type of tumor. The rarity of this tumor is exemplified by the fact that only one or two cases have been reported by any one author, with few exceptions. Carmody[1] reported four cases.

Various theories have been advanced as to the development of osteoma. Among these are: first, the development from the bony diploe[2]; second, the development from cartilaginous arrest[3]; third, the development from a periosteal split[4]; fourth, the development from trauma and suppuration[5]; fifth, the development from ossified polypus[6]; sixth, the development from osteophytes.[7]

It is Dahmann's[7] belief that there is more than one method of origin. Fetissof[8] in his recent analysis believes that there are really only two theories: first, development from embryonal cartilage; and second, development from embryonal periosteum.

Pfeiffer[9] also believes that these tumors are of preosseus origin and that the bony growth results partly from osteoblastic activity and partly by direct metaplasia.

*From the Department of the Oto-Rhino-Laryngology of the University of Tennessee.

We are indebted for the illustrations in this study to Dr. Joseph L. Scianni, artist, University of Tennessee Pathological Institute, and to Miss Dorothy Sturm.

Fig. 1, Case 1. Fig. 2, Case 2.

Fig. 1, Case 1. X-ray showing two distinct areas of increased density.

Fig. 2, Case 2. X-ray, lateral view, showing relative positions of the two masses.

Fetissof's work, which agrees with that of several authors, is quite logical and so convincing that his conclusions are quoted as follows:

1. The growth of the osteoma takes place from the inside outward by the metaplasia of fibrous tissue into bone tissue.

2. The osteoblastic ossificatory process that takes place at the periphery of the osteoma plays a secondary role in the growth of the tumor.

3. The fibrous tissue filling the interstices of spongiose bone is the direct continuation of the periosteum covering the osteoma from the outside.

4. The theory of origin of osteoma from the periosteum split-off at the stage of the development of the organism is most fundamental.

We feel that his observations of the histology of these tumors in the different stages of development warrants acceptance of his conclusions.

Anatomically these tumors are of irregular shape and size, depending upon the rapidity and duration of growth. They may occur in the nasal cavities or any of the accessory sinuses.

The tumor may be one of three varieties: (1) eburnated, or compact and hard; (2) mixed, the periphery compact and central

portion spongy, or (3) spongy, consisting of spongy or cancellous bone.

The usual attachment of osteomas is by a cancellous bony pedicle, while a small percentage are attached by a broad base. A certain number of osteomas are described as being detached from their base and lying loose in the sinus. Dolbeau[10] first described these tumors as necrotic bony balls lying in the frontal sinus similar to a urinary calculus. Tillsmann[11] in his article on dead osteomas of the nose and frontal sinuses says that Otto and Holmes Coote found loose osteomas in the frontal sinus. However he knew that the skulls belonged to Richet, and he placed them in the museum at Dupuytren.

Roux and Jobert[12] tried to remove similar growths during the life of the patient but were unable to do so because they were so firmly attached to the bony walls. After death of the patient there was a cadaveric separation of the growths as is seen on the cadaver when the epiphysis is separated from the diaphysis.

Tillsmann[11] like Dolbeau[10] thinks these tumors originate from cartilaginous arrests. He says there is a spontaneous detachment of the tumor from its base. He has observed a typical case in a woman, 53 years old, who had an osteoma the size of an eagle egg in the frontal sinus and also one of large circumference in the nasal cavity, which was unattached and came from the ethmoid. The latter one was removed.

In both instances an attachment was clearly visible as also was the place of attachment on the wall of the ethmoids and extending into the frontal sinus. Both tumors originated in the ethmoid region. Tillsmann[11] in discussing the manner of detachment gave as a probable explanation that the spongy bone at the point of attachment was destroyed through pressure atrophy. Richet[13] is of the opinion that the tumors were detached through suppuration. Tillsmann[11] said that the mucous membrane covering had degenerated, but that the site of the former stem connection could be observed because of the irregular and more spongy bone. We doubt very much whether the two cases that he described ever had a previous bony attachment.

Tillsmann[11] believes that all osteomas are covered by a mucous membrane while alive but that the membrane becomes destroyed by (1) pressure on the surrounding walls, (2) suppuration, (3) pressure necrosis continued to the pedicle with resultant loss of nutrition, or finally (4) loss of nutrition, the stem having been broken by trauma.

Fig. 3, Case 1. Drawing showing position of the two osteomas resting on the floor of the right frontal sinus with a portion of the floor and anterior wall removed.

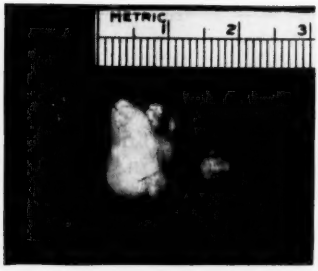


Fig. 4, Case 1. Photograph of the two osteomas showing actual size.

Bornhaut[13] in discussing ten cases of osteoma of the antrum of Highmore stated that in three of them separation occurred by suppuration.

Guthrie[14] stated that the pedicle of osteomas is usually quite small in relation to the size of the tumor and sometimes, apparently as a result of absorption of the bone, consists of fibrous tissue only. It may even undergo complete atrophy, the growth thus becoming free in the cavity. (The so-called "dead osteoma.")

Garrettson[15] stated that there is usually a pedicle which joins the tumor to some part of the sinus and that the pedicle is cancellous in structure. There are cases on record in which this pedicle has become detached, giving rise to the so-called "dead osteoma" which is occasionally found in the cadaver. When detached these tumors become freely movable and form more or less of a foreign body. He also states that the mucosa which covers the projecting tumor into the sinus cavity usually shows polypoid degeneration.

His observation that the mucous membrane on the surface of the osteoma is usually polypoid has not been mentioned to our knowledge by any other author, but in our most recent case this condition was found to exist.

Sewall's[16] case would probably be classed as a loose osteoma. However he states that during the operation as he removed the anterior wall of the sinus he cut into the tumor, the tumor being in perfect apposition to the anterior wall of the sinus at this point. No attachment of the tumor to the sinus wall was found, and he stated that probably none existed.

Several years ago while doing a frontal sinus operation on a fresh cadaver, an osteoma the size of a small hazel nut was found in the left frontal sinus. The growth was lying free in the sinus, and there was no evidence of there ever having been any previous bony attachment or signs of previous infection. The tumor was hard with a smooth surface except for a small roughened area on one side. It is very probable that the tumor received its nourishment through this roughened area.

Adolph Eckert-Moibus[17] mentions fifteen loose or dead osteomas of the nasal accessory sinuses and states that the osteomas may be extruded spontaneously when small or remain as foreign bodies when large.

A word in regard to osteoma of the mastoid which is indeed a rare manifestation. There is a slightly movable type of osteoma in

Fig. 5, Case 1.

Fig. 6, Case 1.

Fig. 5, Case 1. Photomicrograph X100 of the larger tumor showing the irregular pattern of bone with lacunae containing bone cells. Haversian spaces with blood vessels and periosteum to the left. Also demonstrating the roughened surface of attachment to the right.

Fig. 6, Case 1. Photomicrograph X100 of the smaller tumor demonstrating vascular fibrous tissue attachment and periosteal covering. Structure is similar to that of Fig. 5.

this region which is usually situated in a depression of the mastoid bone with an intervening mass of cartilage or connective tissue. MacCuen Smith[18] reported a case of osteoma of the mastoid in which there was an intervening layer of skin instead of cartilage or connective tissue. Emil Amberg[19] reported the only case in the literature of a loose osteoma of the mastoid. A second case of loose osteoma has been seen by us and was removed and studied microscopically. Amberg's case possibly had a pedicle, but in our case there was no bony pedicle, and the tumor was entirely free in a large mastoid cell. Our tumor was slightly roughened on one side from which there was doubtless some vascular supply.

We are firmly convinced that a high percentage of the loose or so-called "dead osteomas" are alive and growing. In our opinion they receive their vascular supply through the small roughened area which has been described. This area is so small that it would usually be overlooked at the time of removal and would only be detected by microscopical studies. This statement is based upon our studies of several loose osteomas including four of our own, which had no visible attachment but appeared to be alive and growing. We believe that Sewall's[16] case was alive and growing with possibly only a small

pedicle, too small to be noticed or recognized grossly. There are other cases that we think are of a similar nature, which have been described as dead osteomas but some of which probably received their nutrition through a small connective tissue pedicle not detected at the time of operation. We repeat for emphasis that many of these osteomas received their nutrition through a small connective tissue pedicle which was not detected at the time of operation.

However, we are quite aware of the fact that it is possible for a small number of the so-called loose osteomas to have a minute bony pedicle in conjunction with the connective tissue pedicle which could be very easily fractured unknowingly during removal of the tumor or prior to this time by trauma or disease.

In one of our cases a connective tissue attachment was demonstrated microscopically which was highly vascular and extended into the body of the tumor. Grossly, however, there was no visible attachment other than a roughened area on one surface which could have been easily overlooked as a source of nutrition.

From our studies we are convinced that all osteomas need not have a bony pedicle or attachment to be alive and growing.

In the living it is possible for this slight attachment as well as the tumors with small bony pedicles to become detached through (1) suppuration, (2) pressure atrophy, (3) trauma. One could readily visualize what would occur in the cadaver to such small vascular connective tissue attachment, this factor alone accounting for the large percentage of loose osteomas being found after death.

Microscopically the tumors are usually covered from the outside with ciliated, columnar epithelium underlying a reticulated submucosa, interspersed with capillaries, round cells and an occasional mucous gland. The periosteal covering is usually of two layers, a thin outer and a fairly thick inner germinative layer which extends into the mass of the tumor. It seems to be the opinion of most writers that the structure found in these new growths does not differ a great deal from normal bone. The results of the microscopical studies of Fetissof's[8] cases show that the osteoblastic ossificatory process plays a minor role in osteoma as compared with the formation through metaplasia from fibrous tissue which is in turn a direct continuation of the periosteum. The spongy portions of the osteoma should be considered its younger and the compact portion its older part.

Fig. 7, Case 2.

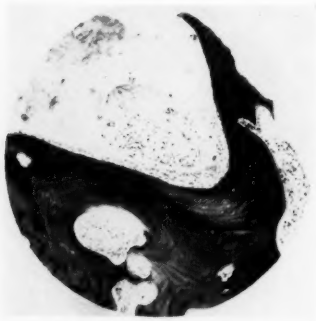


Fig. 8, Case 2.

Fig. 7, Case 2. X-ray showing relative position and size of tumor, also thickened membrane of the frontal, ethmoid and maxillary sinuses.

Fig. 8, Case 2. Drawing showing osteoma in the frontal sinuses and polypoid mucous membrane in the sinus and covering the tumor.

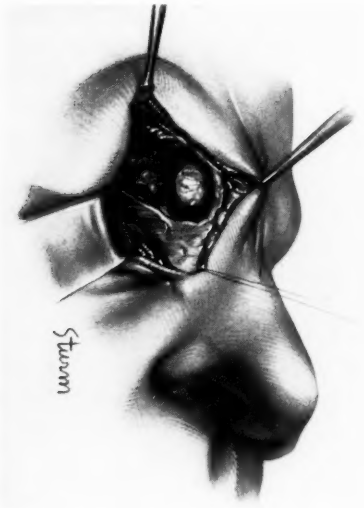


Fig. 9, Case 2. Drawing showing osteoma and mucous membrane removed from sinus and from surface of tumor.

We are not inclined to agree with Fetissof's[8] statement that the osteoblastic ossificatory process plays a minor role as compared to the metaplastic process from fibrous tissue. This is possibly true in cases of bony new growths occurring where bony tissue is not normally found, but where they are found to be a direct continuation of the periosteum it seems that the osteoblastic ossificatory process should play a major role in formation of the tumor.

Rawlins[20] believes that the compact bone differs somewhat from normal bone in that it is very irregular with canaliculi extending in a parallel arrangement near the periphery and irregularly in bundles in the deeper parts and that this compact bone may be surrounded by dense connective tissue which passes directly into the bone, only small areas being found with definite bony lamellae arranged in concentric structure.

In the spongy type there are irregular groups of interlaced trabeculae surrounded by connective tissue growing into the bony framework of the tumor. Fibrous tissue bundles make an irregular network throughout the tumor. Obviously the picture varies somewhat depending upon the age and activity of the cellular structure. According to Fetissof[8] fibroblasts may be seen in the connective tissue bundles, which change into bone through what he terms metaplasia.

In the central portion of these tumors polynucleated giant cells and large single nucleated cells may be seen as well as osteoblasts near the surface of the lamellar structure. As a rule, however, very little activity is seen in this type of new growth because they are such slow growing tumors.

To differentiate osteomas from exostoses (bony hyperplasia-trauma) microscopically, is indeed a delicate task, but we are dealing clinically with two different types of bony overgrowth. In the case of the osteoma we have to deal with a neoplasm having an unlimited growth, displacing adjacent structures, and if not removed in its entirety one apt to recur in a much more rapid form of growth. In exostoses there exists a bony hyperplasia of a more or less diffuse nature produced by some inflammatory change in the periosteum. When this and the inflammatory irritant are removed the exostosis does not tend to recur.

Symptoms, of course, vary with the size and location of the tumor. If it is small and not in a position to obstruct drainage from the sinus in which it lies and if the sinus is free from infection, there will probably be no symptoms. On the other hand if it is

larger, the symptoms will vary with the degree of displacement and site of invasion of normal structures.

X-ray, of course, is our most valuable adjunct to diagnosis, these tumors seldom being diagnosed prior to x-ray studies.

Early surgical removal is advocated because of their malignant tendencies to displace adjacent structures and the complications arising from infection.

Handousa[21] says, "Symptomless osteomata in adults can be safely left alone as they are not likely to grow bigger and give rise to symptoms later or change into sarcomata. Extra-nasal exploration is the method of choice, good access being essential."

CASE REPORTS

CASE 1.— C. R. (colored) female, aged 34, admitted to the John Gaston Hospital December 26, 1939. The patient was admitted for treatment of acute maxillary sinusitis, for which she received repeated antral irrigation. The patient stated that whenever she had a cold she was troubled with severe right-sided frontal headaches. She was discharged January 5, 1940, symptom free, and was advised to return in three weeks for further study.

Routine x-ray studies of the sinuses revealed two areas of increased density in the right frontal sinus (Figs. 1 and 2).

The patient returned on January 31, 1940, and was operated upon under local anesthesia on the first of February. The external approach well below the brow was used. Two bony tumors were encountered resting on the floor of the sinus as the frontal and anterior ethmoids were opened. The smaller tumor was about the size of a pea and the larger, the size of a small hickory nut. The smaller tumor lay more medially near the septum of the frontals, while the larger was lying more laterally, extending well out in the sinus cavity. Neither tumor had a visible attachment. The mucous membrane covering the tumors seemed to be normal. Both masses fell out of the sinus as the inferior wall was very carefully and slowly removed. A complete ethmo-spheno-frontal exenteration was performed. Patient was discharged February 5 to the out-patient clinic for further follow-up.

Pathological Reports: Macroscopic: specimen consists of two small masses. The smaller is irregular, globoid, measuring .5 cm. in diameter. It was apparently attached by a stem-like process. It is grayish-pink and very firm. The largest mass is tongue-shaped and was attached at the small end. It is nodular, grayish-pink and firm and measures 1.5 x 1.3 x .8 cm.

Microscopic: Both masses after decalcification consist of a compact bone with small and large haversian spaces containing delicate fibrous tissue and capillaries. The bone is composed of lacunae with radiating canaliculi. In the lacunae are small bone cells. The pattern is more irregular than normal. The surface except at a small area of attachment is covered by a fibrous tissue periosteum. Diagnosis: Compact osteoma.

CASE 2.—L. T. (colored) male, aged 37, admitted to the John Gaston Hospital April 22, 1940. Patient complained of headaches and stuffiness of the nose of four years' duration. He had the sensation that something was loose in his head which seemed to move from side to side with movements of his head. Examination revealed a polypoid mass in the right naris almost completely obstructing the airway. There was a mucoid discharge in the nasopharynx.

X-ray studies showed haziness of the right frontal, ethmoid, and antrum; also an area of increased density about the size of a hazel nut in the right frontal sinus was noted and a diagnosis of osteoma was made.

The right frontal sinus was opened the following day by the usual external approach with low brow incision. Mucous membrane of the frontal showed considerable polypoid degeneration and a bony mass in the posterior medial aspect of the sinus adjacent to the septum. The mass was firmly attached by a bony pedicle, which was fractured with chisel and mallet. The tumor was covered with polypoid mucous membrane. A complete ethmo-spheno-frontal exenteration was performed. The patient was discharged to the out-patient clinic on April 26.

Pathological Report: Macroscopic: specimen consists of a globoid, firm mass of grayish color, apparently covered by a pink, translucent mucoid membrane. It is attached by a broad base. It measures .7 cm. in diameter. There were submitted also two small, pink, translucent, succulent pieces of tissue measuring about 1.7 x .5 x .2 cm.

Microscopic: the decalcified globoid mass consists of compact bone with lacunae containing small bone cells; radiating from the lacunae are canaliculi. The haversian spaces contain a delicate fibrous tissue and blood capillaries. The pattern is less regular than normal. Most of the outer surface is covered by a delicate periosteum except at the area of attachment.

The two pieces of succulent tissue consist of a vascular fibrous tissue mostly covered by a ciliated columnar epithelium. The one is rich in polymorphonuclears and eosinophiles, the other in polymorphonuclear neutrophiles and large and small mononuclears. Diagnosis: Osteoma and infected fibrous polyps.

BIBLIOGRAPHY

1. Carmody, T. E.: Osteoma of the Nasal Accessory Sinuses. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 44:626 (Sept.), 1935.

2. Arnold: Virchows Osteome des Stirholen. Arch. f. Path. und Anat., 57:145, 1872.

3. Bornhaupt: Ein Fall Von links-seitigen Stirnhohlen Osteom. Arch. fur Klinische Chirurgie, 26:589, 1881.

4. Henrici: Kieferhohlenosteome Medizinische Klinik, 7:432, 1911.

5. Cloquet: Bull. de la Soc. de Chir., 1885.

6. Zucher and Dolbeau: Bull. Acad. de Med., 31, 1886.

7. Dahmann: Uber das Osteom der Nasennebenhohlen. Zeit fur Holenasen Ohrenheilk, 50:261, 1922.

8. Fetissof, A. G.: Pathogenesis of Osteoma of Nasal Accessory Sinuses. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 38:404 (June), 1929.

9. Pfeiffer: A Case of Osteoma of the Frontal Sinus with Perforation of the Cerebral Wall. Zeit f. Ohrenh., 64:223, 1912.

10. Dolbeau: Exostoses du Sinus Frontal. Bullet. de l'Acad. de Med., 31:31, 1886.

11. Tillsmann, H.: Uber todte Osteoma der Nasen und Stirhohle. Archiv. fur Klinische Chirurgie, 32:677, 1885.

12. Roux and Jobert: Archiv. fur Klinische Chirurgie, 32:677-690, 1885 (from Tillsmann).

13. Richet: Bullet. de l'Acad. de Med., 36:564-599, 1871.

14. Guthrie, T.: Case of Osteoma of Septum. Laryng., 45:189, 1930.

15. Garrettson, W. T.: Osteoma of the Frontal Sinus. Arch. Oto-Rhino-Laryng., 5:135, 1927.

16. Sewall, E. C.: Osteoma of the Frontal Sinus. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 27:275, 1918.

17. Eckert-Mobius, Adolph: Guartige Geschwulste der innern Nase u. ihrer Neben Hohlen. Handbuch der Hals-Nasen Ohrenheilkunde by Denker Kahler, 5:107, 1906.

18. Smith, MacCuen: An Unusual Growth of the Mastoid Process: Fibro-Chondro-Osteoma of the Mastoid Antrum. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 15:218, 1906.

19. Amberg, Emil: An Irregular Pearl-Shaped Bone Formation in the Mastoid Process New York Med. J., 92:709, 1910.

20. Rawlins, A. G.: Osteoma of the Maxillary Sinus. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 47:735, 1938.

21. Handousa, A. S.: Nasal Osteomata. J. of Laryng. & Otol., 55:197, 1940.

LXXVII

BLACK TONGUE: REPORT OF TWO CASES*

L. R. MARSHALL, M.D.

BATAVIA

Black tongue is a rather unusual condition. According to Padgett, since its description by Rayer in 1835, only fifty cases have thus far been reported. This estimate appears to be too low. In approximately ten thousand admissions to the hospital with which the writer is associated, only two genuine cases were noted. Not all cases admitted, however, were examined by the otolaryngologist. Black tongue in the human being is not to be confused with the so-called "black tongue" in dogs. The latter is the manifestation of a deficiency disease in dogs, resembling human pellagra, and is in no way related or similar to black tongue in man.

Clinically, the condition appears as a pigmented hypertrophy of the filiform papillae on the dorsum of the tongue. The color may range from a black brown to a brown yellow. The disease is distinguished from ordinary furred tongue by a remarkable hypertrophy of the filiform papillae which gives the dorsum of the tongue a hairy appearance. The papillae may vary in length from one twenty-fifth to three-fourths inch. They can be pulled out without pain or hemorrhage. The hypertrophic process usually begins on the posterior mid-dorsal surface of the tongue and may extend forward until it covers most of the dorsum. Spontaneous recession may take place.

There is no complete agreement as to etiology. A black mold has been cultivated by Cianglinski, Hewelke and Sandiak, but this finding has not been confirmed by others. A yeast-like organism called mucor rhizopodiformis has been isolated by MacFarland, while a streptothrix found by Weidman produced brown pigment in culture. Analysis of most findings indicates that the mold fungi are probably accidental or occasional members of the oral flora. Schech, Brosin and Heidingsfeld believe that the disturbance is simply a pronounced type of hyperkeratosis of the filiform papillae, and the

*Published with permission of the Medical Director, Veterans' Administration, who assumes no responsibility for the opinions expressed.

Fig. 1, Case 1.

Fig. 2, Case 2.

discoloration merely due to an intensification of the normal color of their horny structure as a result of age, drying out and condensation of the superficial epithelial cells. In a few animals such as the cat, giraffe, parrot and certain species of dogs such as the Chinese Chow, a normal bluish-black pigmentation is seen.

Study of all the etiologic possibilities advanced leads to the conclusion that the disturbance consists primarily of a hyperkeratosis of the filiform papillae produced by irritating substances and a secondary deposit of pigment upon and between the papillae from external sources. The pigments which produce the color orginate from external sources such as foods, condiments, stimulants, medicines and from mouth washes containing oxidizing or reducing chemicals or from tannic acid. Pigmentation may result from a chemical reaction between the iron present in the blood and certain sulphur and ammonia compounds resulting from the decomposition of food debris. Iron may react to form iron sulfide or iron tannate. Decomposition of hemoglobin by peroxide of hydrogen may produce pigment.

Histologic study shows that the process is essentially a hyperkeratosis and not an inflammatory reaction.

Subjective symptoms are not common. The tongue may feel dry. There may be salivation or a ticklish sensation on the palate.

Occasionally a patient may complain of gagging. In certain introspective individuals, symptoms become marked when their attention is called to the condition.

The prognosis is good, but relapses occur frequently when treatment is discontinued. As stated before, spontaneous recession may occur, and the condition may disappear completely only to return again.

Treatment in mild cases consists of the application of full strength perovide of hydrogen followed by warm saline solution. In severe cases, a ten to fifteen per cent solution of salicylic acid in equal parts of glycerine and alcohol acts as an efficient keratolytic agent. As a preliminary measure the papillae may be "shaved" down with a razor blade. The application of the foregoing keratolytic agent should be followed by peroxide of hydrogen and saline solution. Oxidizing mouth washes and astringents containing tannic acid should be avoided.

CASE 1.—B. H., white male, aged 52, was admitted for the treatment of neurasthenia. He stated that for the past nine years he had noted a brown furry growth on the dorsum of his tongue. The chief complaint was gagging. He seemed more concerned with its appearance, however, than with the symptom produced.

Examination revealed a hyperkeratosis of the filiform papillae with brown pigmentation. The papillae reached a height of about seven millimeters and gradually tapered down in size toward the middorsum of the tongue. It was possible to pluck out bunches of the papillae without bleeding. Microscopic examination was confirmatory. No bacteriologic studies were made.

Treatment consisted of shaving the papillae down with a safety razor blade and then applying a solution of fifteen per cent salicylic acid in equal parts of alcohol and glycerine. The foregoing solution was rinsed off with hydrogen peroxide followed by normal saline.

As long as treatment was continued the condition could be controlled. Interruption of treatment was promptly followed by relapse. There was no specific factor in his physical condition which could be regarded as definitely etiologic.

CASE 2.—S. D., white male, aged 46. The black furry growth had been present for six years. At times it spontaneously disappeared, only to return. He was an out-patient and declined treat-

ment, inasmuch as it did not trouble him. A clump was pulled out and examined microscopically. The growth consisted of hyperkeratotic papillae.

BATAVIA, N. Y.

BIBLIOGRAPHY

1. Prinz, Herman, and Greenbaum, Sigmund: Diseases of the Mouth and Their Treatment. Lea and Febiger, Philadelphia, 1935.

2. Heidingsfeld, M. L.: J. A. M. A., 55:2117, 1910.

3. Prinz, H.: Dental Cosmos, 65:690, 1923.

4. Weidman, F. D.: Arch. Dermat. and Syph., 18:647, 1928.

5. Wells, C. R., and Cooper, G. W.: U. S. Naval Med. Bulletin, 24:1.

6. Padgett, E. C.: Surgical Diseases of the Mouth and Jaws. W. B. Saunders and Co., 1938.

LXXVIII

MODIFICATION OF THE STEVEN'S RUBBER NASAL BALLOON PACK

WALTER A. FORD, M.D.

SHEBOYGAN, WISCONSIN

The following is a description of some changes and improvements in the Steven's rubber nasal balloon pack. This pack was designed for use in severe nasal hemorrhages in which some sort of postnasal pack is necessary. The air-inflated rubber bag was found to be easily introduced into the nose anteriorly, thereby eliminating the need for applying a postnasal plug through the mouth. The rubber pack, after deflation, is easily removed without damage to the tissues.

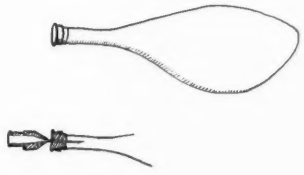


Fig. 1.

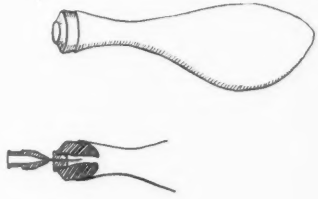


Fig. 2.

The Steven's pack is inflated by hypodermic needle through a plug at the end of a narrow neck. This neck is easily punctured by the needle during inflation and deflation. In fact, extreme care must be exercised to avoid this as, of course, the bag is of no use if it leaks. During inflation, also, the present bag has a tendency to slip back into the nose and down into the nasopharynx. This may be very embarrassing if it happens. Once inflated, the neck of the bag must be securely fastened to a gauze sponge which acts also as a plug for the anterior nasal orifice.

The modification which I have designed does away with the three major objections described above.

(1) The rubber plug (Fig. 2) protects the needle end during inflation and deflation.

(2) It effectively prevents the bag from slipping back into the nasopharynx.

(3) It plugs the nostril to prevent bleeding anteriorly.

In practice the rubber balloon pack is easily inserted into the nose. It is thoroughly deflated and then can easily be rolled into a tight cylinder about cigarette size. It may then be lubricated with K-Y jelly and slipped into the nose. A ten or fifteen cc. all glass Luer syringe may be used to put in the air.

SHEBOYGAN CLINIC.

LXXIX

RETROPHARYNGEAL LIPOMA

FLOYD J. PUTNEY, M.D.

AND

KENNETH E. FRY, M.D.

PHILADELPHIA

Lipomas may develop in any part of the body where adipose tissue is located, yet their occurrence in and around the pharynx is observed very infrequently. Lipomas in this region are usually grouped as pharyngeal growths, which include nasopharyngeal, retropharyngeal, hypopharyngeal, and extralaryngeal tumors together with those arising from the appendages of the pharynx. This designation is not wholly satisfactory, for although retropharyngeal growths are found in the pharynx and are discovered when they produce symptoms referable to respiration or deglutition they originate outside of the pharyngeal cavity and mucous membrane. Retropharyngeal lipomas, therefore, should be classified under a separate anatomic heading from pharyngeal tumors.

In 1927, Figi and Hunt included a case of their own from the Mayo Clinic in a series of 35 collected cases of lipoma of the pharynx and emphasized the need for a more accurate classification. Four years later another case was added by New. Previously Herriman had collected a group of 25 cases from the literature. Pharyngeal lipomas have been infrequently observed in this country, and most of the reports are found in foreign literature. We have listed under retropharyngeal lipomas only those cases in which the tumor mass was situated between the pharynx and the vertebral column. The cases of this type collected from the literature appear in Table I.

The symptoms are produced by an interference with deglutition or respiration. Tumors in this locality are rarely noticed except when the symptoms become marked and the swelling large. A feeling of a "lump in the throat" may be the first indication of any abnormality. Dyspnea, especially in a prone position, is frequently noticed due to the bulging forward of the posterior pharyngeal wall over the aperture of the larynx. Noisy breathing while asleep is a common

TABLE I

Cases of Retropharyngeal Lipoma

NO.	AUTHOR	DATE	AGE	SEX	SIZE	TREATMENT	RESULT	REMARKS
1.	Taylor	1877	4	F.	3 x 3.5 in.	Tracheotomy	Death	Asphyxiation
2.	Roe	1879	7	F.	Large	Peroral removal	Recovery	.....
3.	Milligan	1902	37	F.	Extended from soft palate to hypopharynx	External incision	Recovery	.....
4.	Kenyon	1912	27	F.	10 x 7 x 3 cm.	Peroral removal	Recovery	.....
5.	Rethi	1917	47	F.	120 gm.	Lateral pharyngotomy	Recovery	.....
6.	Pfalz	1918	Aged	M.	6½ x 6 x 4 cm.	.....	Not stated	.....
7.	Tusini	1923			5 x 6 x 9 cm.	External incision	Not stated	Tracheotomy
8.	Hajek	1924	40	F.	15 cm. in length	Lateral pharyngotomy	.....	.....
9.	Faulder and Jefferson	1924	Middle aged	M.	15 cm. in length	External incision	Not stated	.....
10.	Woods	1924	41	M.	Larger than a fist	External incision	Recovery	Laryngotomy for anesthesia
11.	Carrega	1926	60	M.	Enormous	External incision	Recovery	.....
12.	Figi and Hunt	1927	50	M.	7 x 6 x 2.5 cm.	External incision	Recovery	Preliminary tracheotomy
13.	New	1931	51	M.	7 x 5 x 4 cm.	Peroral removal; ligation of external carotid artery	Recovery	Center composed of bone
14.	Patterson	1933	42	M.	7 cm. in length	External incision	Death	Broncho-pneumonia developed after infection of wound by patient
15.	Larsen	1934	71	M.	9 x 7 x 2.5 cm.	External incision	Recovery	.....

complaint. Speech changes such as thickness or indistinctness may be early symptoms. As the tumor increases in size progressive dysphagia develops. Inability to propel a bolus of food beyond the pharynx and lodgement of food at that site are noticed. Weight loss may occur from lack of sufficient nutrition and sleep.

On examination of the pharynx the interval between the soft palate and posterior pharyngeal wall is greatly diminished, and a smooth swelling of the posterior wall can be seen. The swelling appears smooth, non-ulcerated, and may be located in the mid-line but more often predominates on one side. The enlargement may extend from the nasopharynx to the hypopharynx, and a view of the larynx is often obscured by the overhanging mass. On palpation the tumor is moderately firm and compressible. When the tumor is large there is usually a mass present in the neck, more commonly unilateral, with an indefinite outline and soft consistency. Lobulations can rarely be distinguished.

Retropharyngeal lipoma must be differentiated from a malignant tumor or abscess in this locality. A retropharyngeal abscess develops after a short illness and is associated with marked prostration, fever, leucocytosis, and fluctuation. The history of onset, the outline, consistency, and fixation distinguish a benign from a malignant lesion. The diagnosis should be made without the aid of a biopsy. In removing a piece of tissue for histologic examination by the intraoral route the normal pharyngeal mucous membrane has to be incised, and the risk of contamination and infection from the pharynx becomes great. When the tumor is approached externally one should be prepared to remove it entirely as soon as its identity becomes established.

Treatment is surgical extirpation, preferably by an approach through the neck. The technique of removal is relatively easy because the mass is sharply delimited from the surrounding tissue and shells out readily. An incision along the anterior border of the sternomastoid muscle provides adequate exposure. The tumor may extend from the base of the skull to the apex of the lung and may lie in close approximation to the carotid sheath. It is not necessary to ligate the external carotid artery preliminary to operation. The danger of severe hemorrhage is remote if the incision affords a view of the important structures in the neck.

The following patients were admitted to the Jefferson Hospital within a period of 15 months.

Fig. 1. The mass is seen protruding into the pharynx almost occluding it.

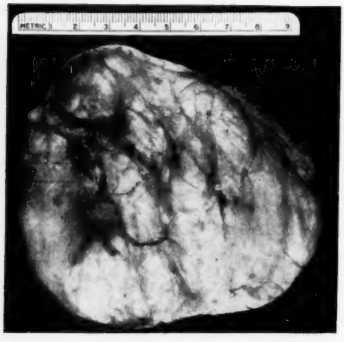


Fig. 2. The lipoma, following its removal.

Fig. 3. Lateral roentgenogram which demonstrates the displacement anteriorly of the larynx and trachea.

REPORT OF CASES

CASE 1.—A white male, aged 57, was admitted to the service of Dr. Thomas A. Shallow, complaining of dysphagia and dyspnea of such severity that he found it necessary to sit up in bed to sleep. Ten years prior to admission the patient noticed a lump just below the angle of the right mandible. After two years the swelling had reached the size of a hen's egg, but was painless. At this time dry foods such as bread and peanuts would stick in his throat. A physician made the diagnosis of a tumor and roentgen ray therapy was given. Forty treatments were given over a period of three years with no noticeable change in the size of the tumor.

For the past three or four years he had had some dysphagia and dyspnea, and the tumor had gradually increased in size. The patient had recently lost his ability to sing.

Physical examination revealed a mass extending from the right mandible to the right clavicle. It extended from the mid-line of the neck to the posterior margin of the right sternomastoid muscle. The tumor lay deep to this muscle, was smooth in outline, not tender, and soft. It could be moved laterally but not in the vertical direction. Pharyngeal examination revealed the mass protruding from the right side into and practically occluding the pharynx. The mass was soft, being almost semi-fluctuant (Fig. 1).

Novocaine one per cent solution was employed as an infiltration anesthetic. This was deemed to be the safest anesthesia as the passing of an intratracheal tube was thought to be ill advised. The incision was made along the full length of the anterior border of the right sternomastoid muscle. Retraction of the muscle laterally revealed a fatty tumor lying under the pretracheal layer of the deep cervical fascia. The tumor extended from the base of the skull to the apex of the right pleural cavity lying between the bodies of the cervical vertebrae and the pharynx. The entire mass was extirpated, the vessels of supply being ligated prior to their division. A small soft rubber tissue drain was placed in the depths of the cavity and the wound closed in layers. The drain was removed on the third postoperative day, and the wound was well healed in one week. The patient's dysphagia disappeared almost immediately and the dyspnea gradually. Fourteen days after operation he was discharged from the hospital with no complaints.

The specimen (Fig. 2) consisted of an irregularly shaped mass of fatty tissue measuring 12 x 7 x 3 cms., and weighing 210 grams. Histologic examination revealed it to be well encapsulated and composed of fat cells with a fibrous tissue reticulum. The diagnosis was lipoma.

CASE 2.—A white male, aged 40, was admitted to the service of Dr. Louis H. Clerf, complaining of swelling of the neck associated with marked shortness of breath, thickness of speech, and inability to sleep. The symptoms dated back two years when speech difficulty was noticed following tonsillectomy. Dyspnea gradually developed so that the patient had to remain in a semi-recumbent position in order to rest. He stated that after sleeping for several hours he would suddenly awaken gasping for air.

Physical examination revealed bilateral fullness of the neck, which was more marked on the right side producing deviation of the trachea to the left. On

inspection of the pharynx a large retropharyngeal tumor was noted. The swelling began about 1 cm. above the soft palate and became progressively larger from above downward, meeting the posterior surface of the epiglottis (Fig. 3). Although the mass was soft and overhanging the larynx, pulsation and fluctuation were absent.

Under intratracheal ether anesthesia a well circumscribed lipoma was exposed through an incision along the anterior border of the right sternomastoid muscle. The tumor extended from the first to the sixth cervical vertebra and was situated between the vertebral column and the pharynx. Separation was not difficult, and the entire growth was removed. The specimen measured 10 x 5½ cm. and on histologic section was composed of fat cells supported by a fibrous tissue reticulum.

Following completion of the operation and removal of the Flagg airway the redundant and edematous pharyngeal mucosa was drawn into the orifice of the larynx with each breath, producing obstructive inspiratory dyspnea. Tracheotomy was necessary to provide an adequate airway. The patient made an uneventful recovery with decannulation on the tenth and discharge from the hospital on the fourteenth postoperative day. Since leaving the hospital the patient has been free of symptoms and has returned to his usual occupation.

The chief problem presented in the surgical removal of retropharyngeal growths is the provision of an airway sufficient to prevent asphyxia during the course of the anesthesia. The safest method of maintaining the patency of the airway during operation is by intratracheal intubation. This is essential if general anesthesia is employed. In one of our cases local anesthesia was used with an intratracheal tube available, while in the other a Flagg intratracheal tube was inserted before the general anesthetic was started.

SUMMARY

Retropharyngeal lipomas produce symptoms only when they grow sufficiently to cause obstruction to respiration or deglutition. Differentiation from retropharyngeal abscess and malignant tumor can usually be accomplished without incision or biopsy. The treatment consists of surgical removal through an external neck incision. Care must be taken to keep the airway patent during the operation, and this can best be done by intratracheal intubation before beginning the anesthetic. Although a larger number of lipomas of the pharynx have been reported, only 15 cases of retropharyngeal lipomas could be found in the literature, to which two cases are added.

JEFFERSON HOSPITAL.

LXXX

ACUTE SUPPURATIVE OTITIS MEDIA: A STATISTICAL STUDY OF 1514 CASES, OF WHICH 896 WERE SURGICAL*

J. H. Maxwell, M.D.

AND

D. H. Brownell, M.D.

Ann Arbor

The many questions which arise during the treatment of any case of acute suppurative otitis media have prompted the following statistical study in which the conditions found in a series of more than 1500 cases of this disease are recorded.

These patients have been observed from the standpoint of attempting to determine what influential factors may exist which alter the course of a case of acute suppurative otitis media and what predetermines either spontaneous resolution, or a suppurative process in the temporal bone demanding surgical intervention. We have also sought to find whether the etiologic background and bacteriologic type of infection decides the path which the disease will follow and whether the general constitutional make-up of the patient, altered perhaps by some chronic systemic disease, changes the direction taken by the infection. What errors of omission or commission on the part of the physician might be responsible for various complications of the middle ear infection, and does the time and type of middle ear drainage play an important part in determining the course of the disease and the prognosis? An attempt has also been made to discover whether myringotomy is less important in infants than in older individuals. All these are questions which arise during the treatment of this disease and this study will attempt to answer them by means of carefully tabulated charts.

Since a very small percentage of all patients with acute suppurative otitis media develop surgical mastoiditis, a study of a large

*Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc.

From the Department of Otolaryngology, University of Michigan Medical School.

number of consecutive cases in one community would yield but few mastoidectomies. The type of case of acute suppurative otitis media seen in the University of Michigan Hospital lends itself particularly well to statistical study because the percentage needing surgical intervention is high, due to the fact that most of the patients are referred to the clinic because of the suspected presence of a complication of the acute middle ear infection. As over one-half of the cases observed demanded operation, it was felt that here was an opportunity to analyze various factors in the etiologic background and preoperative management which might have had some bearing on the development of complications. Children who developed their middle ear infections while in the hospital under treatment for other conditions served to some extent as a control group, comprising the very large percentage of non-operated cases.

Because of the lack of opportunity to culture a significant number of all groups of cases at the onset of the infection, the bacteriological data has been omitted from this discussion although about 3000 records were reviewed.

From these 3000 records, 1514 were selected for the statistical analysis because detailed information of the onset, course and termination of the otitis media was available. Among these 1514 cases, 1166 represented unilateral infection in a like number of patients, and 348 demonstrated bilateral ear infection in half the number of actual patients. This required tabulating of one patient as two cases when bilateral otitis media was present has disturbed chiefly the recovery and death data. Thus some of the mortality rates are materially raised, because a death has been recorded for each of two separate cases of otitis media occurring simultaneously in the same patient.

Statistical study of any large series of clinical cases is greatly facilitated by the use of a punch-card coding system and sorting machine, with which we studied these 1500 cases in great detail. A punch-card was prepared for each, containing data on sixty-five factors and recording up to nine subdivisions of each factor. Actually, 3860 correlations were made, the sorting and tabulating machine making it possible to find the totals for each separate factor and also any conceivable combination of factors. Eleven tables were prepared from these correlations, and the charts accompanying this discussion represent an attempt to portray the significant data which they contain.

In the charts the cases are classified as septic and non-septic. The term sepsis is used broadly to indicate a general clinical picture and does not signify an otitic sepsis in every instance. In most of those patients with sepsis who did not have a surgical mastoiditis, the otitis media was an incident in the course of another severe infectious process, such as pneumonia or one of the acute exanthematous fevers. We have considered as septic those patients who, during the course of an otitis media, have developed leucocytosis and intermittent fever rising to 103 degrees F. or higher at least once daily and who have continued to manifest such evidence of illness without abatement for five days or longer. By adopting these criteria of sepsis we have excluded from the septic group those acutely ill patients who demonstrated a gradually subsiding fever and leucocytosis following tympanic drainage.

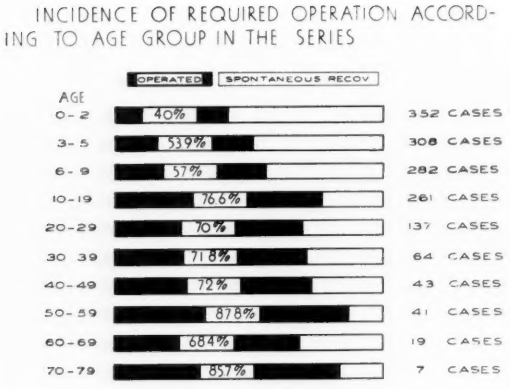


Chart I.

CHART I.—The cases of otitis media in this series are divided according to age groups in the above chart. In each age group the percentage of patients requiring mastoidectomy is indicated. The lower incidence of surgical mastoiditis in infants and young children is attributable in some measure to the fact that many cases of acute otitis media complicate epidemic upper respiratory infections among children in the hospital under treatment for other conditions. Thus in this group a larger number of routine cases is encountered which demonstrate the usual tendency to spontaneous resolution. In the older age groups most of the cases seen in this clinic are referred because of a suspected complication of the otitis media.

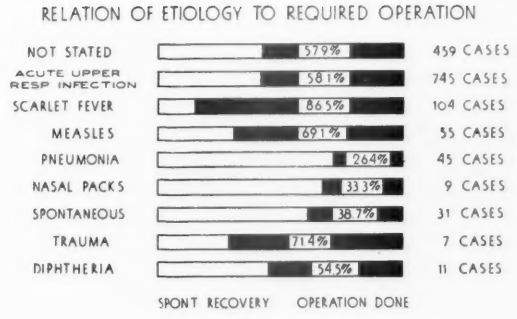


Chart II.

CHART II.—In the cases studied, a high incidence of required operations is noted in individuals with otitis media of scarlet fever and measles origin. Noteworthy is a small percentage of cases of acute suppurative otitis media associated with pneumonia in which mastoidectomy was necessary. The only possible significance of the 71.4 per cent of traumatic cases operated upon lies in the fact that the seven cases listed resulted either from swimming accidents or skull fractures. A rather large number of fractures of the tympanic membrane from dry blows upon the ear have been seen in the clinic but are not recorded here because they were not followed in detail until complete healing of the drum membrane occurred.

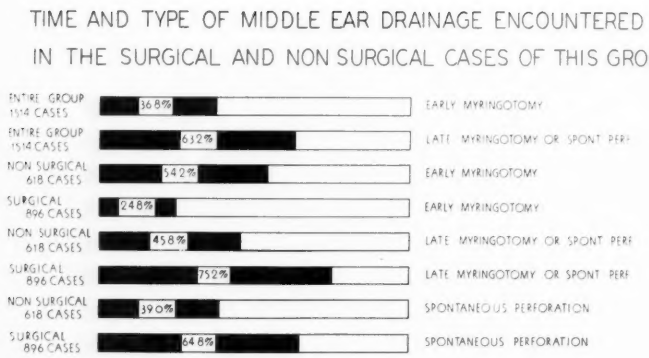


Chart III.

CHART III.—The majority of patients with acute suppurative otitis media seen in this clinic did not have the benefit of early drainage of the infected tympanum. Recorded here are all of the cases in this group divided according to the time and type of drainage of the middle ear. Drainage established before three days of more or less continuous severe earache is regarded as early, while drainage after three days of symptoms, which from the history suggested actual suppuration, is considered to be late.

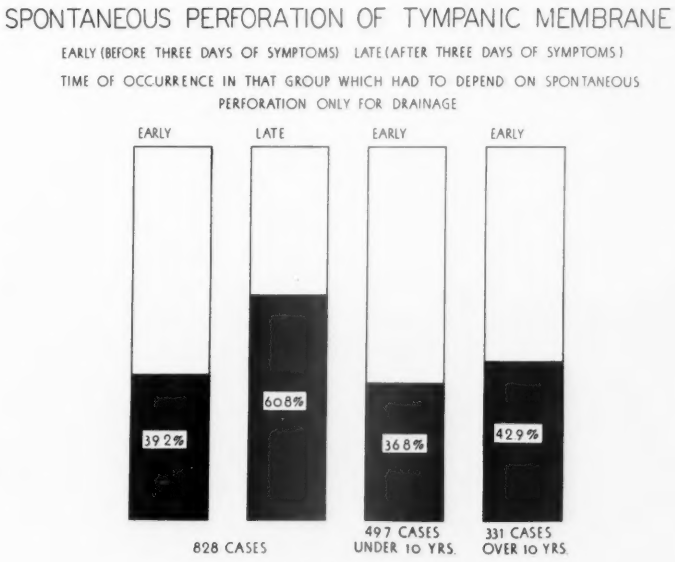


Chart IV.

CHART IV.—There were 828 of the 1514 cases which had to depend upon spontaneous perforation alone for drainage of the middle ear. It was found that the majority of these spontaneous perforations occurred after three days of symptoms. In other words less than 40 per cent of patients having spontaneous perforation of the tympanic membrane had this relief before three days of symptoms of acute suppurative otitis media. In separating these patients into different age groups it is found that there is no special tendency for spontaneous perforation to occur earlier in children. The little difference noted in the time of spontaneous perforation of the drum membrane in children under ten years of age might suggest that there is a decreased tendency for early spontaneous perforation in young individuals.

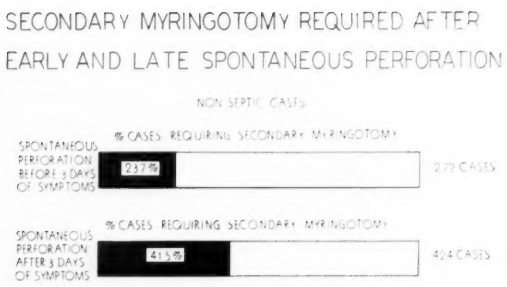


Chart V.

CHART V.—The adequacy of drainage established by spontaneous perforation of the tympanic membrane is very doubtful when we consider, as noted in the above chart, that many of these patients demanded secondary myringotomy because of the obviously inadequate perforation noted at the time of examination. It was interesting to find that a larger number of secondary myringotomies was demanded in the cases giving a history of late spontaneous perforation. Perhaps a larger opening in the tympanic membrane is produced when this structure is thin enough to rupture early in the course of the disease.

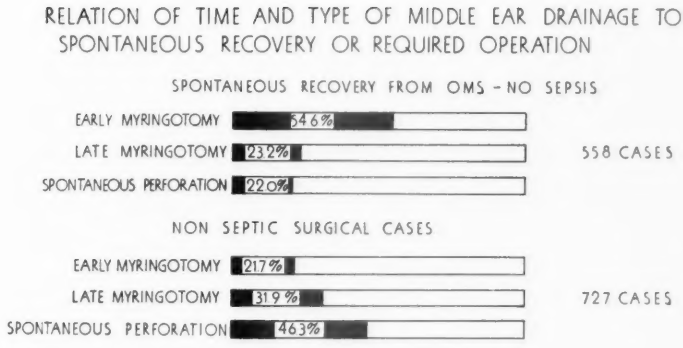


Chart VI.

CHART VI.—A study of the relation of the time and type of middle ear drainage to spontaneous recovery or required operation demonstrates that the greatest number of patients who had early myringotomy is found in that group which recovered spontaneously. Of the 558 cases in this group, there were 54.6 per cent who had early myringotomy, while the remainder had delayed drainage either by late myringotomy or spontaneous perforation alone. The converse situation is noted in the group of operated cases in which the vast majority did not have the benefit of early drainage of the tympanum.

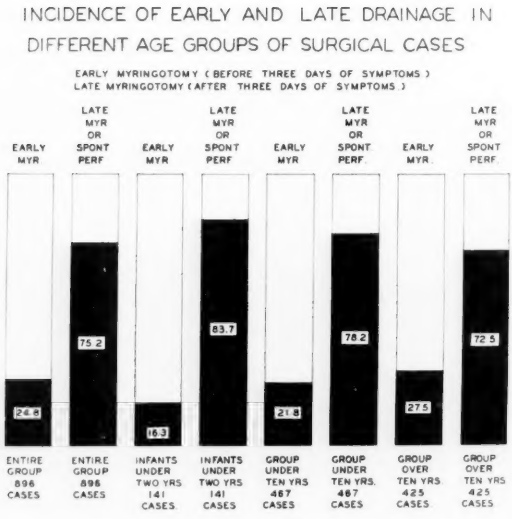

INCIDENCE OF EARLY AND LATE DRAINAGE IN DIFFERENT AGE GROUPS OF SURGICAL CASES
EARLY MYRINGOTOMY (BEFORE THREE DAYS OF SYMPTOMS)
LATE MYRINGOTOMY (AFTER THREE DAYS OF SYMPTOMS)
EARLY MYR
LATE MYR OR SPONT PERF
24.8
75.2
16.3
83.7
21.8
78.2
27.5
72.5
ENTIRE GROUP 896 CASES
ENTIRE GROUP 896 CASES
INFANTS UNDER TWO YRS. 141 CASES
INFANTS UNDER TWO YRS. 141 CASES
GROUP UNDER TEN YRS. 467 CASES
GROUP UNDER TEN YRS. 467 CASES
GROUP OVER TEN YRS. 425 CASES
GROUP OVER TEN YRS. 425 CASES

Chart VII.

CHART VII.—Not only is late myringotomy or spontaneous perforation of the tympanic membrane noted much more frequently in the entire group of surgical cases than is early myringotomy, but it is also observed that there is no essential variation in any of the age groups. There is a slight but distinct tendency to find a smaller percentage of patients in the older age group of surgical cases giving a history of late drainage of the tympanum.

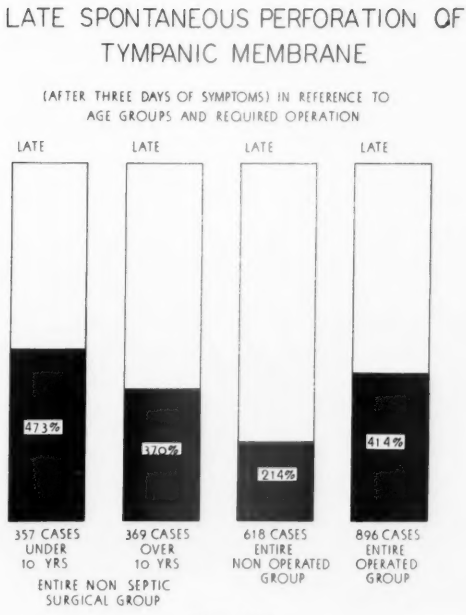


Chart VIII.

CHART VIII.—As was noted in a previous chart, spontaneous perforation of the tympanic membrane is more apt to occur late than early. In the cases studied, late spontaneous perforation was found much less frequently in those individuals recovering spontaneously from their infection than in those who required operation. It is of interest to note the higher incidence of late spontaneous perforation of the membrane in children under ten years of age.

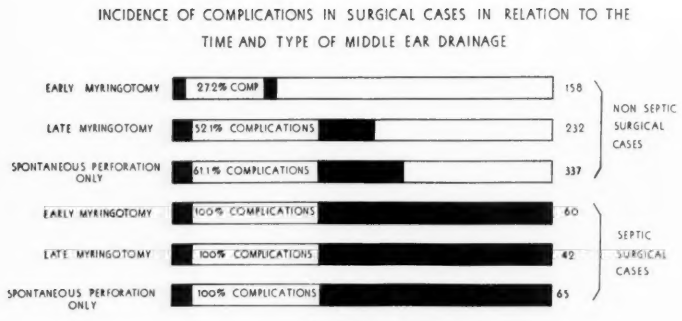


Chart IX.

CHART IX.—In the non-septic surgical cases in this group there is a direct relationship between the number of complications and delayed drainage of the tympanum. This is not noted in the septic cases, which throughout this study have shown little tendency to be influenced by early therapeutic procedures. The complications include subperiosteal abscess, Bezold's abscess, facial paralysis, labyrinthitis, parasinus abscess, sigmoid sinus thrombosis, brain abscess, meningitis, pneumonia, pyelitis, erysipelas, septic joint and endocarditis.

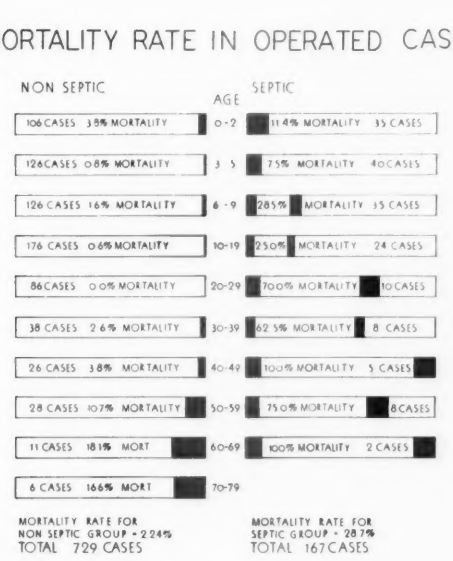


Chart X.

CHART X.—The mortality rate in the operated cases in this group is shown in the above table. There is noted a definite tendency for the mortality rate to increase with increasing age in both the non-septic and the septic groups of surgical patients. In the non-septic group a sharp rise is noted after the age of 60 years, while in the septic cases the rise in mortality rate is found after the age of 20 years.

In the non-septic cases in infants less than two years of age, the listed mortality rate of 3.8 per cent is inaccurate. In this study, as was described in the introduction, the number of cases represent the number of infected ears rather than the number of individuals. As before mentioned, this system was used in order to facilitate punch-card coding. In the 106 non-septic surgical cases less than two years of age there were two deaths. Both of these children had bilateral acute suppurative otitis media, one being complicated by pulmonary tuberculosis, dehydration and diarrhea, and the other by pyelitis and infectious diarrhea, which conditions were ascribed in each case to be the principle causes of death. Thus,

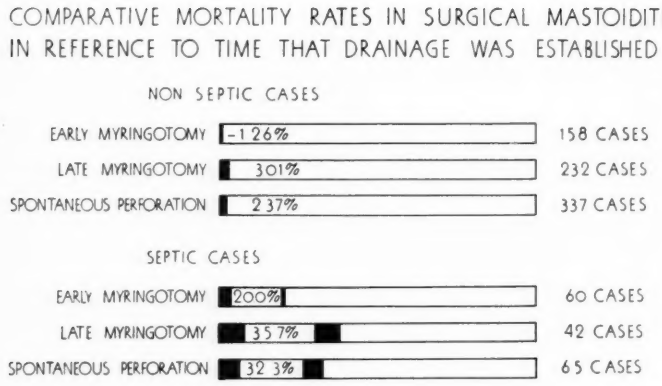


Chart XI.

actually in this group we can report a mortality rate of 1.9 per cent. The septic cases do not represent cases of otitic sepsis alone. In most of those septic patients who did not require operation, the otitis media was an incident in the course of another disease such as pneumonia, meningitis, an acute eruptive fever, etc., which was responsible for the sepsis.

CHART XI.—An inquiry into the possible relationship between mortality rate and time and type of drainage of the infected tympanum reveals the fact that in all surgical cases, whether septic or non-septic, the mortality rate was much higher in those individuals who did not have the benefit of early drainage. In fact the mortality rate nearly doubled in those cases having late myringotomy or depending upon spontaneous perforation alone. In 36 of the 48 deaths recorded in septic surgical patients there had been late drainage of the middle ear.

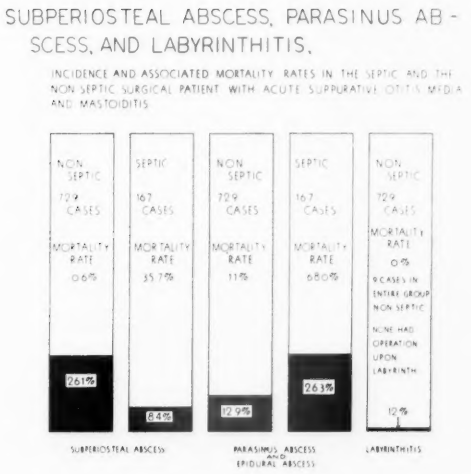


Chart XII.

CHART XII.—The incidence and associated mortality rate in cases demonstrating subperiosteal abscess, parasinus abscess or labyrinthitis as complications of acute suppurative otitis media and mastoiditis are indicated in the above chart.

Subperiosteal abscess showed a relatively more frequent occurrence in the non-septic patient than in the septic patient. In the latter the mortality rate was much higher.

Parasinus abscess and epidural abscess were encountered more frequently in the septic than in the non-septic patient. In the former there was found a much higher mortality rate.

In the entire group of 729 non-septic surgical cases evidence of acute labyrinthitis was encountered in nine instances. None of these patients succumbed, and none required an operation upon the labyrinth.

EFFECT OF ASSOCIATED CONSTITUTIONAL DISEASE UPON THE MORTALITY RATE IN CASES OF ACUTE SUPPURATIVE OTITIS MEDIA

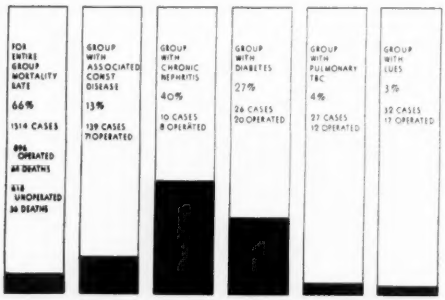


Chart XIII.

CHART XIII.—Associated constitutional disease, such as chronic nephritis and diabetes, seems to predispose the patient with acute suppurative otitis media to a septic course. A septic course was noted in 15.5 per cent of the entire series and in 22.3 per cent of patients with associated constitutional disease.

In the patients with associated constitutional disease the mortality rate was much higher than the average for the whole group. In the recorded cases diabetes and chronic nephritis were the most influential in raising the mortality rate.

The mortality rate of 6.6 per cent for the entire group included, of course, deaths in a large number of septic patients suffering from an acute suppurative otitis media which contributed but little if anything toward the fatal outcome.

SUMMARY

We have coded 1514 cases of acute suppurative otitis media in which detailed information was available from the beginning of the infection to its termination, and have analyzed them by means of the punch-card system. From a large number of correlations made, those revealing data of statistical value have been tabulated.

Graphic charts are included to illustrate the more significant of these tabulations.

Spontaneous perforation of the tympanic membrane was more apt to occur after three days of symptoms of otitis media than earlier in the disease.

There was no tendency for spontaneous perforation of the tympanic membrane to occur earlier in children than in older individuals.

Fewer secondary myringotomies were required after early spontaneous perforation of the tympanic membrane than after a late appearance of this condition.

The majority of cases of acute surgical mastoiditis had late drainage of the acute suppurative otitis media, whether by late myringotomy or by spontaneous perforation.

Early adequate drainage in cases of acute suppurative otitis media was encountered more frequently in those cases recovering spontaneously than in those requiring mastoidectomy.

The time and type of surgical drainage seemed, from this study, to bear a signficant relationship to the course of the otitis media.

There was a definite relationship between the incidence of complications in surgical cases and the time and type of middle ear drainage.

In the non-septic surgical cases of mastoiditis there was a sharp rise in the mortality rate after the age of 60 years, while in the septic ones the mortality rate was more than doubled after the age of 20 years.

Associated constitutional disease, such as chronic nephritis and diabetes, seemed to predispose the patient with an acute suppurative otitis media to a septic course and was definitely influential in increasing the mortality rate.

A study of comparative mortality rates in surgical mastoiditis demonstrated that a higher rate was associated with late drainage of the tympanum.

CONCLUSIONS

The time and type of middle ear drainage in cases of acute suppurative otitis media are influential factors in determining the course of the disease.

Spontaneous perforation of the tympanic membrane, which has no tendency to occur earlier or to be more adequate in children than in older individuals, is more apt to occur after three days of otitis media than before and often furnishes an inadequate drainage opening.

Early adequate surgical drainage of the tympanum is necessary in cases of acute suppurative otitis media to preclude the deleterious effects of pressure retention of pus within the middle ear.

In non-septic patients with suppurative otitis media, delayed drainage of the tympanum is conducive to the development of mastoiditis, increases the incidence of regional and remote suppurative complications of the disease and raises the mortality rate.

Furthermore, the septic cases of otitis media, in which the stormy course and also many of the complications seem to be uninfluenced by early therapeutic procedures, show a remarkable increase in mortality rate when tympanic drainage is delayed.

Although it is not contended that the pressure factor is the only one of importance in the production of mastoiditis and its complications, its role is one of major significance.

201 S. Main St.

LXXXI

PEMPHIGUS LOCALIZED IN THE MOUTH

E. I. Matis, M.D.

Kaunas, Lithuania

Isolated pemphigus in the mouth is very rare and presents many peculiarities in its development and much difficulty in diagnosis. Such cases of pemphigus localized in the mouth require the attention of an otolaryngologist whom, according to New and O'Leary,[1] patients generally consult first, especially if signs on the external body surface are absent. The otolaryngologist has difficulty not only because he has had little experience with the disease but also because the localization of it in the mouth is uncommon, and many typical pemphigus symptoms are absent. Pemphigus is usually localized on the skin of the body where characteristic signs are visible.

In the past the term "pemphigus" has been applied somewhat loosely to a large number of skin affections characterized by the formation of bullae. It is now generally agreed to reserve the term for four varieties of bulbous eruptions: (1) pemphigus acutus malignus; (2) pemphigus vulgaris; (3) pemphigus foliaceous, and (4) pemphigus vegetans (Robert MacKenna[2]). Among these forms there is a considerable difference in prognosis and course.

Pemphigus acutus malignus is a rare disease, acute at the onset with elevation of temperature, and the mortality rate is high—60 to 70 per cent.

Pemphigus vulgaris runs a more chronic course and tends to relapse after intervals of complete or partial disappearance of all lesions. Prognosis of pemphigus vulgaris is easier than that of the other forms.

Pemphigus foliaceous is a more serious form of the previous one mentioned.

Pemphigus vegetans is a rare and serious form which affects adults only. The disease usually ends fatally within a few months. In pemphigus foliaceous and pemphigus vegetans, the mouth is often affected.

Regarding the etiology of pemphigus no definite knowledge is available. According to many writers on the subject, pemphigus is

caused by neurotropic disturbances, but this hypothesis has not been proved. The establishment of a specific virus has not yet been successful, and various experiments concerning the infection of one individual from another have failed. Bacteriological examinations have not given any important results, and it is not unlikely that the cause will yet be found to depend on some toxin absorbed either from the gastrointestinal tract or from some septic from elsewhere in the body (MacKenna[2]).

Pemphigus is primarily a disease of the skin, but the mucosa may be affected, for example, that of the mouth, lips, pharynx and larynx. In rare cases pemphigus may be localized in the nose, which gives rise to nasal obstruction and nose bleeding (Farlow[3]). Pemphigus may begin as a tonsillar manifestation (Dintenfass, Greenbaum[4]).

According to Schäffer[5] the spread of pemphigus to the mouth makes the course of the disease considerably worse.

If, according to Figi,[6] manifestations of pemphigus are not infrequently encountered in the mouth, then the presence of skin lesions facilitates the diagnosis even if the mucosal lesions appear a little prior to their development. Where the disease appears only in the mouth, diagnosis is considerably more difficult. The author had under observation five cases of pemphigus in which it was for a long time localized in the mouth and pharynx. In some of these cases diagnosis could not be made for a long time, and only during the course of the illness was the true nature of it revealed. With one exception, a woman of twenty, all were middle-aged women. They all appeared during the course of the same half year but came from various parts of the country so that no general cause can be suggested. It is important, in the author's opinion, to stress the fact that typical bullae on non-inflammatory bases were extremely rarely detected. Also a careful observation of the patients for several days did not always result in finding these bullae. A very important fact is that the bullae, which are the principal signs of pemphigus, are extremely thin walled, and being continually exposed to mechanical injury in the pharyngo-oral-cavity they rupture easily within a few hours (Weiss[7]). "Following rupture, the flaccid pellicle sloughs off leaving a superficial, painful ulcer which promptly develops a marked secondary inflammatory reaction" (Figi[6]). The mucosa appears to be only slightly wounded, and around these wounds the mucous membrane is of a slightly whitish-gray color. The margins are often occupied by whitish shreds, the remains of former bullae roofs. The color of these shreds may alter and become a dark grayish-brown for

instance, while a foul smell emanates from the mouth. At the commencement these wounds are situated only in one part of the mouth or lips, but as the disease progresses they spread and then appear in the pharynx. The patient complains of piercing, burning pains in the throat, which become worse, and then of difficulty in swallowing. From this moment his general condition declines. He begins to eat less and less, trying to avoid foods that might cause irritation, and quickly weakens and gets thin. The condition is now very serious, but it becomes still more so when the disease spreads to the larynx. The patient suffers severe pain and feels a desire to cough up the membrane which remains after the bullae have burst. However, such attempts to cough cause severe pain. As will be observed in the case reports, the disease spreads deeper into the eustachian tube, and the patient suffers great pain on swallowing and coughing, and dies of pneumonia. It is presumed that such a gradual development of the disease is typical of pemphigus. However in many cases intervals are observed, and sometimes the bullae disappear, giving the impression that the disease is passing away, but from the standpoint of prognosis one must be very careful. In his cases the author has noted intermissions in the disease with the result that the illness progresses, passing into the deeper parts of the eustachian tube. Even when the development of the illness is watched most carefully it is almost impossible to detect the bullae. In the pharynx they very quickly burst. It is presumed that the end is brought about to a great extent by mechanical causes, by coughing and difficulty in swallowing. In only two of the cases observed by the author were there solitary bullae on the skin of the body. It must be stressed that the appearance of such bullae must be carefully watched, as they may considerably facilitate early diagnosis. The prognosis in all cases was bad, and death resulted.

The differential diagnosis of pemphigus of the mouth may be very difficult as the affection may be confused with erythema multiforme which also produces bullous lesions in the mouth. However, according to Figi, this condition is, as a rule, readily differentiated by the history of similar attacks which disappear spontaneously, the seasonal incidence in the autumn and spring, and by the multiformity and distribution of the lesions, especially on the extremities. Other bullous eruptions may be produced by the local application of many drugs, but these are usually found on easily accessible parts of the body, and it is often possible to discover in the lesions some traces of the irritant with which they have been provoked. Various drugs also cause bullous lesions in the mouth, for example, mercury, anti-

pyrine, bromides, barbiturates, and acetanilids. At the beginning of pemphigus in the mouth, it is sometimes difficult to distinguish it from the more severe forms of stomatitis. Lesions on the buccal mucous membrane may suggest diphtheria or syphilis.

Treatment of pemphigus of the mouth is an extremely thankless one. Even remedies which are considered most successful—germanin and blood transfusions—gave no decisive results in many of the author's cases. Also the use of various drugs such as arsenic, strychnine and sulphur had no influence on the disease. The local application of various drugs as mouthwashes or ointments was of little benefit. In stronger solution they irritated the mucosa of the mouth, and in weaker they had no influence on the development of the eruptions. The duodenal sound introduced for the purpose of better nourishment was also very painful for the patient to bear. In the cases the author has dealt with, he found that the disease improves and also passes away spontaneously without the influence of drugs, but nothing can be done to avoid its reappearance.

REPORT OF CASES

CASE 1.—Mrs. F. Z., aged 58 years. On December 12, 1934, she appeared at the hospital complaining of pain in the mouth. Five weeks before this, pain had appeared and had gradually become worse until she was unable to speak or swallow. On examination, the whole of the mouth, tongue and pharynx were found to be covered with a white membrane under which was a raw, red, moist layer. The patient could not swallow and had to be fed through a duodenal sound. It was only two days later that a characteristic bulla was found in the pharynx. The patient was treated with germanin given intravenously and had two blood transfusions. After the first of the transfusions there was a remarkable improvement, but of short duration. Later on the deep parts of the pharynx and larynx were also covered with membrane resulting from the bursting of bullae. It was extremely difficult to give the patient nourishment. On January 29, 1935, the patient's temperature rose, and pneumonia was diagnosed. Medium sized bullae were now visible on the patient's back, and on February fifth she died of general weakness.

CASE 2 was that of a woman of about 50 years of age, and the disease ran a similar course to that mentioned above.

CASES 3 and 4.—In the other two, both women of 43 and 48 years of age, respectively, the course was more chronic. There were intermissions. In these two cases a few bullae were detected on the external body.

CASE 5.—Miss X, 20 years of age. She first of all became ill with a high temperature and eruptions on the mucosa of the mouth. These signs were so indefinite that exact diagnosis was not possible. The illness lasted seven weeks. As the signs were in the front part of the mouth, it was thought that it was stomatitis. There were no bullae visibile in the mouth, and the illness spread further. Finally a bulla the size of a cherry was found on the elbow, but there were

no others. Of course the appearance of this one bulla was sufficient to confirm the diagnosis made at the onset. The patient recovered without any serious consequences, but ten weeks later there was an outbreak of typical pemphigus on the external body, complicated by eruptions in the mouth, from which death resulted. This case is interesting because it shows how difficult it sometimes is to diagnose pemphigus of the mouth.

CONCLUSIONS

In conclusion the author desires to point out the following:

1. The illness may be localized in the mouth only.

2. The appearance of bullae on the external body may come about only towards the end of the illness.

3. Bullae in the mouth, pharynx and larynx may not be detected because they quickly burst. This fact sometimes considerably complicates diagnosis.

4. Any spread of the illness from the anterior parts of the mouth to the pharynx and larynx does much to facilitate diagnosis because this spread is not common in many mouth diseases.

VASANO 16 D. G—VE 4.

REFERENCES

1. New and O'Leary, P. A.: Pemphigus from the Laryngologist's Standpoint. Arch. Otolaryng., 1:617, 1925.

2. MacKenna, R.: Diseases of the Skin. London, 1937.

3. Farlow, cited by Weiss,[7] p. 75.

4. Dintenfass, H., and Greenbaum, S.: Pemphigus as a Beginning Tonsillar Manifestation. Laryngoscope, 35:758, 1925.

5. Schäffer, I.: Die Therapie der Haut und venerischen Krankheiten. P. 226, Urban-Schwarzenberg, Berlin-Vienna, 1916.

6. Figi, F. A.: Dermatologic Conditions of the Ear, Nose and Throat. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 48:81 (March), 1939.

7. Weiss, W.: About Chronic Pemphigus of the Upper Air Passages: A Case Which Led to a Severe Laryngeal Stenosis. Medical Vademecum for Rhinologists by B. Lewis, p. 73, Vienna, 1922.

LXXXII

CHRONIC HOARSENESS: REPORT OF THREE HUNDRED CONSECUTIVE CASES*

J. C. Damitz, M.D.

AND

J. Lewis Dill, M.D.

Detroit

Chronic hoarseness may be classified not as a clinical entity, but as a symptom which designates interference or impairment of the normal function of phonation. It is a quite common complaint occurring during the life of almost every individual, any time from infancy to senility. Jackson and Coates[1] in their textbook enumerate more than fifty etiological factors which have been reiterated and stressed in many excellent reviews. We are attempting here to review the literature and to discuss chronic hoarseness as this entity presents itself to the specialist in an average clinic. Our cases have been analyzed as to incidence of the etiological factors and the clinical importance of the duration of the symptom.

The pathology of chronic hoarseness varies in each individual patient. Watkyn-Thomas[7] relates, "Since any condition which roughens the cord produces hoarseness it is obvious how vitally important this symptom is, for any condition includes a little mucus at one end of the scale to carcinoma of the cord at the other." Daily[9] mentions chronic non-specific laryngitis as the most common cause of chronic hoarseness. This clinical entity is invariably due to an unhealthy condition higher in the respiratory passages and is secondary not only to chronic infection of the teeth, tonsils, nose and sinuses, but results also from excesses of tobacco and alcohol (McCready,[10] Pryor,[11] Meyers,[12] Turner[13] and Negus[14]).

Just as pathology of the upper respiratory tract may cause an involvement of the larynx, so also may chronic infection below the vocal cords, as bronchiectasis, lung abscess and bronchial asthma, initiate and prolong a chronic laryngitis by bathing its structures in an irritating discharge with resulting local irritation. Oaks, Merrill

*From the Department of Otolaryngology, Henry Ford Hospital, Detroit.

and Oaks[15] discuss chronic hoarseness without any demonstrable local pathology and reflexly secondary to a chronic ethmoiditis. When searching for the exciting cause of chronic laryngitis, one must realize that external irritants, injury, and general systemic conditions may be the responsible factors. Hirsch[16] enumerates trauma, blows, stab and gunshot wounds, foreign bodies, inhalation of hot, dry, dusty air, stone and metal dust, irritating vapors of chemical substances, both in industry and in war. A thorough physical examination may disclose one or several conditions as the exciting cause and may reveal such diversified conditions as chronic nephritis, diabetes mellitus, Hodgkins' disease as described by Cooke,[17] angioneurotic edema, herpes and beri-beri as discussed by Matsui.[18]

A weakness or paralysis of one or both of the vocal cords is generally noted to be the second most common cause of chronic hoarseness, and this may be due to either a peripheral or a central lesion. The recurrent laryngeal nerves are the most vulnerable and frequently are injured or traumatized during a thyroid operation. Hirsch[16] states that only the crico-arytenoideus posticus may be paralyzed following a thyroidectomy, and hoarseness may not result. If the course of the recurrent laryngeal nerves is considered, it becomes obvious that many local conditions can and do account for many paralyses. Arnold,[19] Lederer,[20] LeJeune[21] and Waugh[8] enumerate such local conditions as trauma of the neck, thyroid enlargement, cervical glands, esophageal carcinoma, mediastinal glands, thickened pleura, thoracic tumors, aortic aneurysms, enlarged heart, dilatation of the left auricle and pericardial effusion. In the articles of Lischkoff,[2] Daily,[9] Woodward[22] and Ridout[23] central lesions which may produce hoarseness are discussed, and mention is made of otitis media with jugular thrombosis, deep cerebellopontine angle tumors, encephalitis lethargica, multiple sclerosis, general paresis of the insane, post-diphtheria, pneumonia, syringomyelia, anterior poliomyelitis, bulbar lesions and toxic neuritis as from lead poisoning.

Benign new growths produce hoarseness as an early symptom in their development. These growths include singers' nodules, discussed by Levbarg[24] and Voorhees,[25] papillomata, fibroma, pachydermia laryngis, chondroma, lipoma, angioma or varix as reported by Robb,[26] and amyloid tumors discussed by Kramer.[27] These are included in the numerous articles of several authors, New and Erich[28] and Jackson, Coates, Jackson,[1] MacKenty,[29] Cunning,[30] Tilley[27] and Daily.[9] Embryonal and blood or lymph retention cysts have been discussed by Decherd.[32] Chevalier Jackson[33] and the late J. E. MacKenty[29] stress the importance of biopsies before any new growth is

considered benign. This becomes more significant when one considers that the possibility of a premalignant change may be present in what otherwise appears to be a benign new growth.

Hoarseness in which a local pathological lesion is evident must always bring to mind the triad of syphilis, tuberculosis and malignancy as the direct cause. Any one of this triad or any combination of these may coexist in the larynx, as noted by MacKenty,[29] Lederer,[20] Woodward[22] and Tucker.[34] Syphilis in its protean manifestations may produce dysphonia in numerous ways, varying from a gumma locally to locomotor ataxia centrally. Where recognition of the etiology is delayed irreparable damage may result locally due to ulceration, infiltration and fixation of the arytenoid cartilages. In any one or a combination of the above mentioned triad, the true vocal cords may be destroyed, whereupon the phonatory function may be assumed by the plicae ventricularis or what has been termed by Jackson and Jackson[35] as "Dysphonia Plicae Ventricularis."

Tuberculosis of the larynx may not produce hoarseness because of the variance of location of the pathological process (Dworetzky[36]). Wagers[37] quotes Patterson, resident physician at White Haven, Pennsylvania sanitarium, who states that of 239 tuberculosis in-patients 82 gave a history of hoarseness upon admission and 49 had definite laryngeal disease. Although hoarseness may not always be present in laryngeal tuberculosis, it may be the first and only symptom of a more generalized infection. Sir St. Clair Thompson[38] states that the larynx is involved in 4.8 per cent of early pulmonary tuberculosis and nearly 50 per cent of the advanced cases.

Carcinoma, "the other end of the scale," produces hoarseness as the earliest and most constant symptom. The extreme importance of the early recognition of this pathological entity cannot be overemphasized, for if this were true there would be few deaths from carcinoma of the larynx. This has been stressed by such authors as Jackson and Jackson,[39] Watson-Williams,[40] Woodward,[22] Powers,[41] Waugh,[8] LeJeune,[42] Watkyn-Thomas,[7] Arnold,[19] Jones[43] and Imperatori.[44] Early intrinsic carcinoma of the larynx offers a better prognosis than it does in any other part of the body because of the lymphatic arrangement; however, the cancer once having gained the regional cervical lymph glands, the prognosis becomes hopeless. Jackson and Jackson[35] state that 98 per cent of cases of intrinsic carcinoma start in or very close to the vocal cords while about 2 per

cent start in regions so remote that hoarseness comes later, but even these cases are rarely incurable by laryngectomy.

Local lesions whether inflammatory, benign, malignant or paralytic may not be present, and when no known lesion exists to account for hoarseness, it must be realized that functional disturbance may be the basis of the symptom.

A study of the last 300 consecutive cases of chronic hoarseness seen in the out-patient department of the Henry Ford Hospital was undertaken from the point of view of etiology and duration of this symptom. No survey of the prognosis, treatment or final outcome of the patient was made.

TABLE I

TABLE OF ETIOLOGY

(1)	Vocal cord paralysis	102 cases
(2)	Chronic non-specific laryngitis	100 "
(3)	Benign neoplasms	41 "
(4)	Carcinoma of larynx	25 "
(5)	Tuberculosis of larynx	23 "
(6)	Syphilis of larynx	1 "
(7)	Miscellaneous group	8 "

In this study, vocal cord paralysis was the chief cause of hoarseness, accounting for 102 cases, or approximately 34 per cent; chronic non-specific laryngitis followed closely, of which there were 100 cases, or 33 per cent. Two-thirds of the cases in this series were due to vocal cord paralysis and chronic non-specific laryngitis, being about equally distributed. In the remaining third hoarseness was produced by benign neoplasms in 41 cases, or 14 per cent; carcinoma, 25 cases, or 8-1/3 per cent; laryngeal tuberculosis, 23 cases, or 7-2/3 per cent. Carcinoma and tuberculosis were about equal in their occurrence, and the two combined occurred slightly more frequently than the benign neoplasms. The remaining cases were due to gumma of the epiglottis, one case, and a miscellaneous group of eight cases.

TABLE II

ETIOLOGICAL FACTORS IN VOCAL CORD PARALYSIS—102 CASES

(1)	Post-thyroidectomy	56 cases
(2)	Non-thyroid cases	46 "
	a. Pulmonary tbc.	5 "
	b. Cardiac failure	3 "
	c. Cervical gland abscess	3 "
	d. Bronchogenic carcinoma	3 "
	e. Metastatic adenocarcinoma	2 "
	f. Psychoneuroses	3 "
	g. Thyroid carcinoma	1 "
	h. Gastric carcinoma with metastases	1 "
	i. Postoperative removal salivary gland	1 "
	j. Multiple sclerosis	1 "
	k. Post-pneumonia	1 "
	l. Post-influenza	1 "
	m. Syringomyelia	1 "
	n. Mediastinal carcinoma	1 "
	o. Gunshot section of recurrent laryngeal nerve	1 "
	p. Myesthenia gravis	1 "
	q. Reidles struma	1 "
	r. Esophageal carcinoma	1 "
	s. Pulmonary carcinoma	1 "
	t. Hodgkins' disease	1 "
	u. Questionable etiology	13 "

TABLE III

DURATION OF HOARSENESS

CASES OF VOCAL CORD PARALYSIS

Post-thyroidectomy group average	3½ years
Post-thyroidectomy group varied	3 days to 11 years
Non-thyroidectomy group average	2½ months
Non-thyroidectomy group varied	1 week to 20 years
Non-thyroidectomy group of cases	
1 to 4 weeks	15 cases
1 to 6 months	13 "
6 to 12 months	8 "
1 to 10 years	4 "
10 to 20 years	4 "
Undetermined	2 "

An analysis of the 102 cases of vocal cord paralysis revealed that 56 followed thyroid surgery and the remaining 46 were produced by a variety of conditions. Diseases of the heart or lungs were responsible in eight cases; carcinoma of related structure such as mediastinum, esophagus, cervical glands and thyroid in 11; local injury or infection in six; lesions of central nervous system in six; post-infectious diseases, as diphtheria, etc., in two; and a questionable etiology in 13 patients. Hoarseness was present as a symptom in all cases of vocal cord paralysis, except in 15 patients following thyroid surgery.

TABLE IV

(A) Chronic Non-Specific Laryngitis—100 Cases

(1)	Chronic purulent maxillary sinusitis	21 cases
(2)	Chronic nasopharyngitis	17 "
(3)	Chronic spheno-ethmoiditis	16 "
(4)	Chronic tonsillitis	12 "
(5)	Chronic productive bronchitis	10 "
(6)	Nasal allergy	8 "
(7)	Chronic frontal sinusitis	1 "
(8)	Chronic atrophic rhinitis	2 "
(9)	Irritants as tobacco, etc.	2 "
(10)	Undetermined etiology	11 "

(B) Duration of Chronic Hoarseness

Cases of Non-Specific Laryngitis

1 to 30 days	37 cases
1 to 6 months	39 "
6 to 12 months	14 "
1 to 6 years	10 "
Average duration	8½ weeks

Chronic infection of the accessory nasal sinuses, either alone or associated with allergy was noted as the etiological factor in over 60 per cent of the 100 cases of chronic non-specific laryngitis. Chronic infection of the bronchial tree or tonsils was the causative factor in the remainder of these cases, except for eleven in whom the etiology was not determined and two in which the laryngitis was secondary to chronic irritation from excess of tobacco. The average duration of hoarseness was eight and a half weeks although in the majority of cases the patients admitted a previous consultation with their fam-

ily physician. Thirty-seven patients had noticed their hoarseness for 1 to 30 days; 39, from 1 to 6 months, 14, from 6 to 12 months, and 10 patients persisted with the symptoms from 1 to 6 years before consulting the clinic.

TABLE V

DURATION OF HOARSENESS

CASES OF BENIGN NEOPLASMS—41 CASES

Average duration of hoarseness	2 years
Hoarseness varied from	2 weeks to 5 years
Average duration and family physician	6½ months
Average duration before seen at Henry Ford Hospital:	
0 to 6 months	13 cases
1 to 5 years	14 "
3 to 10 years	5 "
10 years and over	5 "

TABLE VI

DURATION OF HOARSENESS

CASES OF MALIGNANT NEOPLASMS—25 CASES

Average duration before seen at H.F.H.	9½ months
1 to 30 days	2 cases
1 to 4 months	8 "
4 to 8 months	4 "
8 to 18 months	5 "
18 months and over	6 "

Benign and malignant neoplasms produced hoarseness in every case of our series and together accounted for 22 per cent of this group of patients. As would be expected, benign neoplasms were more frequent than malignant growths in the proportion of two to one. The average duration of hoarseness in the benign cases of our series was almost two years, but in the malignant cases the symptoms of hoarseness were present on the average of nine and a half months. Nearly an equal number of patients consulted our clinic before 18 months; however when the symptoms persisted longer, more patients with a benign lesion were seen than those in whom a malignant new growth was found.

TABLE VII

DURATION OF HOARSENESS

CASES OF TUBERCULOSIS OF LARYNX—23 CASES

Average duration of hoarseness and family physician	4½ weeks
Average duration of hoarseness at Henry Ford Hospital	2⅓ months
1 to 30 days	5 cases
1 to 6 months	11 "
6 to 12 months	3 "
1 year and over	2 "

In our hospital all patients with pulmonary tuberculosis are examined at frequent intervals for laryngeal involvement, and on admission every patient has a laryngeal examination. However in this group of tuberculous laryngitis hoarseness was present for an average of two months before the larynx was seen initially. Five cases were seen with hoarseness of less than 30 days' duration, while only two patients had hoarseness of one year's duration. There were two patients with tuberculous involvement of the larynx with no complaint of hoarseness.

TABLE VIII

DURATION OF HOARSENESS BEFORE ADEQUATE DIAGNOSIS AND TREATMENT INSTITUTED

(1)	Chronic non-specific laryngitis series	8½ weeks
(2)	Vocal paralysis (post-thyroidectomy) series	3½ years
(3)	Vocal paralysis (otherwise)	2⅓ months
(4)	Benign neoplasm	2 years
(5)	Malignant neoplasm	9½ months
(6)	Tuberculosis	2⅓ months

A glance at the preceding chart reveals a marked discrepancy in the average time that patients with different clinical entities appeared for examination, e.g., chronic non-specific laryngitis, eight and a half weeks, and benign new growths for two years. This may be partially explained by the fact that the underlying etiology of most of the cases of chronic non-specific laryngitis brought the patient to the physician earlier than did the isolated symptom of hoarse-

ness. It was noted that several of the patients with benign new growths had known of their condition for years but contrary to professional advice on several occasions preferred to leave "well enough alone." Many of the patients with early carcinoma of the larynx had been treated for nasal and throat infections for weeks and months. Some, of course, presented a more rapid, progressive hoarseness as well as associated symptoms of dysphagia, hemoptysis and aphonia, while others had seen other physicians but failed to heed advice given to them. By the preceding facts we have attempted to explain the average of nine and a half months before this group of patients consulted our clinic. The average of two and a third months for the cases of tuberculous laryngitis may be accredited to the fact that all patients with pulmonary tuberculosis are examined frequently for laryngeal involvement, and a laryngeal examination is given to all patients upon admission.

Hoarseness is still neglected by patient and physician, which may be due to the fact that it so often accompanies and subsides with the common cold or that the patient, as well as the physician, hopes the trouble will "clear up by tomorrow." If the patient were seen early with proper diagnosis and treatment, there would be little necessity of further writing on the subject; however as has been noted in our experience this is far from being true. This is no doubt the experience of every member of the profession. Krieger[6] states that diagnosis should be made by direct or indirect inspection of the larynx or both. Also bacteriological studies of the larynx, physical examination including complete blood counts and chemistry, blood and spinal Wassermann, tuberculin studies, esophagoscopy, bronchoscopy and biopsies should be carried out according to the individual requirements of the patient. Although hoarseness is frequently a trivial matter, it nowise entitles one to treat it as such until proven so, for in such conditions as syphilis, tuberculosis, pre-malignant and malignant neoplasms, irreparable damage ensues if diagnosis and treatment are not instituted early. Daily[9] comments "that in early tuberculous lesions of the larynx, the percentage of cures is one out of every four, and could be more with earlier diagnosis. Some observers believe that carcinoma of the larynx is rare; however Woodward[22] quotes, "Statistics show that approximately 6,000 deaths a year result from it in this country and that 18,000 persons are afflicted with it." In our series, carcinoma and tuberculosis, about equal in occurrence, accounted for about 16 per cent of the cases and continued with their symptom of hoarseness for months until it was too late for treatment to produce the maximum in results.

SUMMARY

1. In our series the occurrence of vocal cord paralysis and chronic non-specific laryngitis were about equal and together accounted for 67 per cent of the cases.

2. There was an unusually close ratio, 41 to 25, of benign to malignant new growths.

3. Carcinoma and tuberculosis were about equal in their occurrence, the former being found in 25 patients, the latter occurring in 23.

4. Syphilis was found to be the etiology only once in 300 patients with chronic hoarseness. It is much more rare in our clinic than reports would indicate.

5. A small group of two per cent were classified as miscellaneous and included cases of psychoneuroses, myasthenia gravis, hysteria and psychopathic personality.

6. The group of patients in our series continued with their symptom of hoarseness for a varying but prolonged period before seeking relief, the average duration of which was as follows: vocal cord paralysis (non-thyroid), two and a half months; chronic non-specific laryngitis, eight and a half weeks; benign neoplasms, two years; malignant neoplasms, nine and a half months; tuberculosis, two and a third months.

In conclusion we must re-emphasize the extreme importance of further educating the public and the profession to the paramount significance of chronic hoarseness as a potentially dangerous symptom. To the profession it is our duty to utilize every means available to reach an early diagnosis and institute treatment.

HENRY FORD HOSPITAL.

BIBLIOGRAPHY

1. Jackson, C., Coates, G. M., and Jackson, C. L.: Nose, Throat and Ear and Their Diseases. W. B. Saunders Co., 1930.

2. Lischkoff, M. A.: Hoarseness. J. Fla. M. A., 22:263 (Dec.), 1935.

3. Friedman, E. D.: Neurological Aspects of Hoarseness. N. Y. State J. Med., 34:48 (Jan.), 1934.

4. Furstenberg, A. C.: An Anatomical and Clinical Study of Central Lesions Producing Paralysis of the Larynx. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:39 (March), 1937.

5. Carpenter, E. W., and Carpenter, Wm. M.: Chronic Hoarseness. S. C. M. A., 33:287 (Dec.), 1937.

6. Krieger, W. A.: Etiology, Symptoms, and Diagnosis of Inflammatory Type of Hoarseness. N. Y. State J. Med., 34:47 (Jan.), 1934.

7. Watkyn-Thomas, F. W.: Hoarseness. Practitioner, 137:681 (Nov.), 1936.

8. Waugh, Justin M.: Significance of Chronic Hoarseness in Adults. Proc. Inter-State Postgrad. M. Assemb., Atlanta, Oct. 15-19, 1928, pp. 522-525.

9. Daily, Louis: Hoarseness: A Danger Signal. South. M. J., 24:1041 (Dec.), 1931.

10. McCready, J. H.: Hoarseness: Its Relation to Local and General Diseases. Pa. M. J., 21:523 (May), 1918.

11. Pryor, W. R.: Practical Consideration of Alterations of Voice. Ky. Med. J., 32:415 (Aug.), 1934.

12. Meyers, E. L.: Hoarseness Caused by Thyroarytenoid Interni Paresis with Symptoms Simulating Acute Pulmonary Tuberculosis Due to a Sinus Infection. Laryngoscope, 29:720 (Dec.), 1919.

13. Turner, A. Logan: Diseases of the Nose, Throat and Ear, 2nd Ed. Wm. Wood and Co., Baltimore, 1927.

14. Negus, V. E.: Diagnosis and Treatment of Hoarseness. M. J. Australia, 2:591 (Oct.), 1933.

15. Oaks, L. W., Merrill, H. G., and Oaks, L. E.: Recurrent Hoarseness and Aphonia Without Demonstrable Laryngeal Pathology. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:1079 (Dec.), 1932.

16. Hirsch, Caesar: Hoarseness: What It Means to Patient and Physician. M. Rec., 141:36 (Jan.), 1935.

17. Cooke, R. G.: Hodgkin's Disease with Unusual Onset. Brit. M. J., 1:308 (Feb.), 1931.

18. Matsui, Taro: Hoarseness of Infants and Examination into its Causes. Laryngoscope, 37:867 (Dec.), 1927.

19. Arnold, H. L.: Neglected Conditions of the Eye, Ear, Nose and Throat. New Orleans M. & S. J., 87:4, 1934.

20. Lederer, F. L.: Diseases of the Ear, Nose and Throat. F. A. Davis and Co., Philadelphia, 1938.

21. LeJeune, F. E.: Hoarseness: Its Significance; Motion Picture Demonstration. New Orleans M. & S. J., 87:5 (July), 1934.

22. Woodward, F. D.: Importance of Hoarseness as Early Symptom of Laryngeal Malignancy. West Virginia M. J., 34:211 (May), 1938.

23. Ridout, C. A.: Aphonia and Hoarseness. Brit. J. J., 1:393 (Mar.), 1929.

24. Levbarg, J. J.: Hoarseness, Especially as Affecting Singers. N. Y. Med. J., 107:744 (April), 1918.

25. Voorhees, I. W.: Non-Surgical Treatment of Aphonia. N. Y. State J. Med., 34:53 (Jan.), 1934.

26. Robb, J. M.: Varix of the Vocal Cord. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 47:522 (June), 1938.

27. Kramer, Rudolph, and Som, M. L.: Local Tumor-Like Deposits of Amyloid in Larynx; Report of Case with Review of Literature. Arch. Otolaryng., 21:324 (March), 1935.

28. New, G. B., and Erich, J. B.: Benign Tumors of Larynx—Study of 722 Cases. Tr. Sect. Laryng., Otol. & Rhin., A. M. A., pp. 17-96, 1938.

29. MacKenty, J. E.: Malignant Disease of the Larynx. Arch. Otolaryng., 20:297 (Sept.), 1934.

30. Cunning, D. S.: Benign Neoplasms of Larynx. N. Y. State J. Med., 34:36 (Jan.), 1934.

31. Tilley, Herbert: Chronic Hoarseness: Its Clinical Significance, Diagnosis and Treatment. M. J. Australia, 2:169 (Aug.), 1933.

32. Decherd, H. B.: Extra-Laryngeal Blood-Cyst in Seven-Weeks Baby. Tex. State J. Med., 19:41 (May), 1923.

33. Jackson, Chevalier, and Jackson, C. L.: Malignant Disease of the Larynx: Its Treatment by Laryngofissure and Laryngectomy. Am. J. Surg., 30:3 (Oct.), 1935.

34. Tucker, Gabriel: A Case of Chronic Hoarseness (Ulcerative Laryngitis) Showing Tuberculosis and Cancer in Same Lesion. J. A. M. A., 96:1572 (May), 1931.

35. Jackson, Chevalier, and Jackson, C. L.: Dysphonia Plicae Ventricularis. Arch. Otolaryng., 21:157 (Feb.), 1935.

36. Dworetzky, J. P.: Modern Concepts of Laryngeal Tuberculosis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 47:481 (June), 1938.

37. Wagers, A. J.: Clinical Significance of Hoarseness. Pa. M. J., 40:14 (Oct.), 1936.

38. Thomson, St. Clair: Diseases of the Nose and Throat. D. Appleton, N. Y., 3d ed., 1926.

39. Jackson, Chevalier, and Jackson, Chevalier L.: The Larynx and its Diseases. W. B. Saunders, Philadelphia, 1937.

40. Watson-Williams, P.: Clinical Import of Hoarseness in Relation to General Practice: With Special Reference to Hoarseness as an Early Symptom of Intrinsic Laryngeal Cancer. Practitioner, 108:153 (March), 1922.

41. Powers, H. W.: Hoarseness. J. Kan. M. Soc., 39:413 (Oct.), 1938.

42. LeJeune, E.: The Significance of Hoarseness. Tex. State J. Med., 33:513 (Nov.), 1937.

43. Jones, D. H., Tuberculous, Malignant and Syphilitic Types of Hoarseness. N. Y. State J. Med., 34:59 (Jan.), 1934.

44. Imperatori, C. J.: Indications for Thyrotomy and Laryngectomy in Carcinoma of Larynx. N. Y. State J. Med., 34:51 (Jan.), 1934.

45. Glushak, L.: Dysphonia Spastica. Laryngoscope, 38:273 (April), 1928.

LXXXIII

ROLE OF OXALIC ACID AND CERTAIN RELATED DICARBOXLYIC ACIDS IN TREATMENT AND CONTROL OF HEMORRHAGE*

ARTHUR STEINBERG
H. I. SEGAL, M.D.
AND
H. M. PARRIS, M.D.

PHILADELPHIA

For over five years one of us (A. S.) has been striving to obtain a hemostatic capable of being administered hypodermically or introduced directly into the circulation, which would rapidly accelerate the rate of coagulation of the blood. Early in the work it was possible to demonstrate that extracts of various plants could be prepared which would accomplish this result when used on experimental animals. Some of these plant sources were shepherd's purse, wood sorrel, sugar beets, oxalis, rhubarb and alfalfa. The extracts varied in their degree of potency as well as toxicity. One of the best preparations was obtained by utilizing shepherd's purse (capsella bursa pastoris). By administering this extract intravenously to rabbits it was possible to produce a marked reduction in the clotting time within an interval of fifteen to thirty minutes. This effect was found to persist from one to six hours (Chart I). On the basis of these experiments a method of assay was devised in which a clotting unit was defined as the minimum amount of extract necessary to produce an average of 50 per cent reduction in coagulation time per kilogram of body weight in three rabbits within 30 minutes of intravenous injection.

The extracts were found to be relatively non-toxic when administered to rats, guinea pigs and rabbits, and they did not produce hemolysis. They were free from protein and contained no alkaloids, and there was therefore no danger of anaphylaxis or habit formation. The plant extract was then administered to normal human subjects, with results comparable to those observed in rabbits. An attempt

*Read at the Forty-Sixth Annual Meeting of the American Laryngological, Rhinological and Otological Society, New York, June 7, 1940.

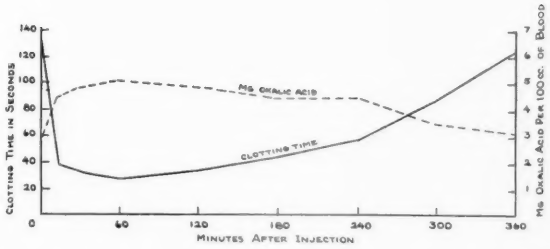


Chart I. Duration of effect of intravenous injection of the plant extract on the clotting time and the oxalic acid content of the blood of rabbits.

was made to limit the metabolic factors which might influence the rate of coagulation. The procedure utilized for the determination of the coagulation time was as follows: Blood was obtained from the vein in a clean, dry syringe; without removal of the needle the blood was allowed to flow into 1 mm. standard bore capillary tubes. If the blood was not obtained with the first puncture the needle was changed to avoid the error due to any tissue juices which had been expressed. A stopwatch was used, and the coagulation time was taken from the moment the blood began to flow into the tube until, when the tube was broken at intervals of approximately 1 cm., a strand of fibrin connected the two broken ends of the tube. The blood was allowed to flow back and forth in the tube during this procedure. It was found desirable to keep the hands close together and supported when breaking the tube, in order to avoid destroying the strand of fibrin. After this test has been performed often, the experimental error can be reduced by reason of its constancy. This test was always performed by the same person, before and after the administration of the drug to preserve the constancy of the error. The normal values ranged from four to six minutes by this method of determination.

Because of the lack of precedent for intravenous administration of a hemostatic it was at first difficult to obtain clinical material. However, it was soon possible to test this preparation on moribund patients in whose cases other measures had failed to control the hemorrhage. Fortunately the results obtained in these cases were such as to make available additional clinical material. Physicians began using this hemostatic for postpartum hemorrhages, for hematemesis, for bleeding resulting from peptic ulcers, for epistaxis and for vari-

ous hemorrhagic dyscrasias. Over a period of four or five years several thousand patients with these types of hemorrhage were treated. The results for the most part were gratifying. In certain cases it was difficult to decide whether the hemorrhage had ceased spontaneously or as a result of the hemostatic used. In several cases of bleeding associated with hemophilia, leukemia, epistaxis or hemoptysis there was little or no response. Extensive hematologic studies have been conducted in order to account for this variation in response of similar types of hemorrhage. No satisfactory explanation has yet been obtained.

An instance of the formation of a thrombus was not observed in several thousand patients treated. No untoward general or local reactions were seen, even in patients treated over a long period. The material was readily excreted by the kidney without producing any damage to the tissue.[1] It has been administered with expedience as an emergency measure, with no special precautions. The response in most cases was rapid, occurring within thirty minutes.

TABLE I

EFFECT OF OXALIC ACID ON COAGULATION TIME IN RABBITS

C.T. Normal: Seconds	Dose, Mg.	C.T. 15 Min. Reduction, Seconds	C.T. 30 Min. Reduction, Seconds	C.T. 60 Min. Reduction, Seconds
153.2	1	132.4	102	93.4
208.6	2	64	42	55
125.8	5	62	34	80
112.4	10	42.8	34.4	52.9
242.2	50	171.4	65	167

C.T., clotting time.

It was found that the activity of the extract was associated with the presence of oxalic acid. When extracts were freed of this substance relatively little hemostatic action remained. By utilizing solutions of chemically pure oxalic acid it was possible to simulate to a large extent the results obtained with the plant extracts (Table I). Furthermore, a large group of available dibasic acids were tested and found to be variously effective biologically in increasing the rate of coagulation. By making solutions of these acids in various combinations it was possible to obtain preparations which would act rapidly

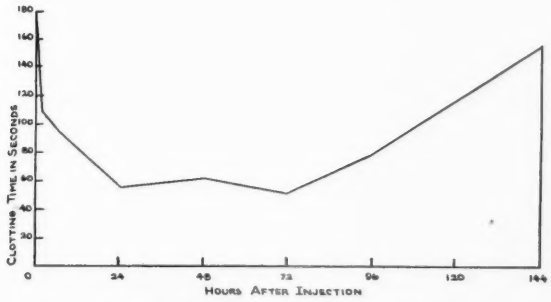


Chart II. Prolonged effect of the ethyl ester of oxalic acid on the coagulation time.

or slowly and have a more or less sustained effect. A preparation of the ethyl ester of oxalic acid was found to produce a reduction in clotting time which was maintained from one to several days (Chart II). Because of its oil solvent it could be administered only intramuscularly. However, when it was used as an adjunct to the aqueous preparation it was possible to obtain an effect both rapid and sustained.

A preliminary report in which many of these data were revealed and in which it was shown that there appeared to be a relationship between the oxalic acid content of the blood and coagulation time (Chart III) was presented by one of us (A. S.) in December 1938.[1] This was followed by a clinical report by Milliken[2] on the use of this hemostatic in transurethral prostatic resection. He stated that the drug is safe and that its use greatly facilitates this operation, gives a clear field in which to operate and saves the time that is otherwise used for coagulating current. Subsequently a report outlining further experimental data was presented by Steinberg and Brown.[3] One of us (A. S.) devised a method for the determination of the oxalic acid content of the blood based on a modification of Suzuki's method,[4] which depends on selective precipitation of oxalic acid by cerium chloride, conversion of the resultant cerium oxalate to oxalic acid and titration with potassium permanganate.

By this method normal values for oxalic acid in the blood of human beings were established at from 5 to 7.5 mg. per hundred cubic centimeters. A correlative trend was manifested between the coagulation time and the oxalic acid level of the blood, as determined by the method outlined at the end of the article. By the same procedure

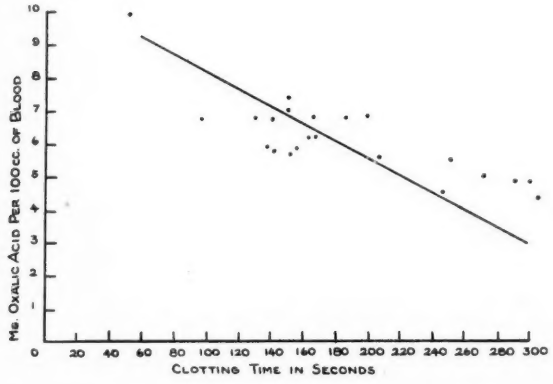


Chart III. Relation of coagulation time to the oxalic acid content of blood.

it was also shown that many body fluids and tissues contain appreciable amounts of oxalic acid (Table II). It has been shown by Ersner, Myers and Ersner[5] that saliva has a hemostatic action. It is possible that this effect was due in part to the oxalic acid content of this material.

TABLE II

Oxalic Acid Content of Body Fluids and Tissues

Substance	Oxalic Acid Content, Mg./100 cc.
Maternal milk	8 — 12.5
Cord blood	7 — 17.0
Placental extract	5 — 8.3
Saliva	10 — 30.0
Bile	15 — 20.0

A series of studies on rabbits was carried out in an attempt to show whether the oxalic acid level of the blood increased when the coagulation time was lowered by small doses of oxalic acid. From Chart IV it may be seen that a diminution in clotting time was attended by a rise in the oxalic acid level of the blood and that the increase in the oxalic acid of the blood was not accounted for by the actual addition of this acid, for the resultant oxalic acid content of

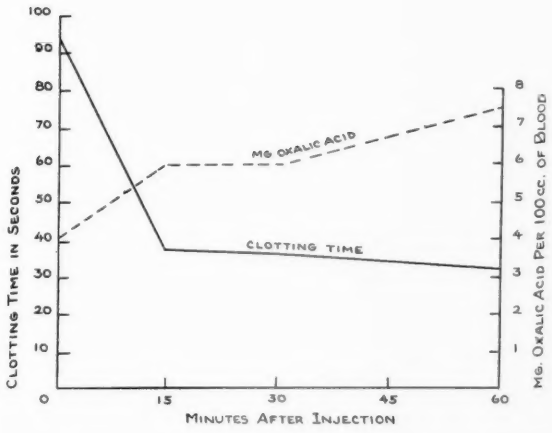


Chart IV. Effect of intravenous administration of oxalic acid (10 mg.) on clotting time and on the oxalic acid content of blood.

the blood was greater than the original value plus the amount added. This response was subsequently demonstrated in human subjects (Table III). There was also observed a transitory decrease in the blood calcium in both animals and human beings. The role of calcium in the coagulation of blood was first shown by Hammersten.[6] Horne[7] showed that addition of an excess of calcium to the blood in a test tube would retard coagulation and that a sample of blood so treated could be caused to clot by the addition of oxalates in sufficient quantity to react with the excess calcium. It was thus observed that in vitro and in vivo experiments are not comparable and that the concentration of the substances used will determine the results obtained. Similarly it was shown that the administration of an excessive amount of oxalic acid to animals will cause a depletion in calcium with a prolongation of clotting time and eventual death, whereas the administration of small amounts of oxalic acid will reduce the time required for coagulation even in hirudinized animals. Hewitt and Pickering[8] showed that the administration of small doses of sodium oxalate in animals was attended by increased coagulability of the blood. Recently it was found that these hemostatic agents would cause a disintegration of the platelets, with the liberation of thromboplastin. A decrease in the prothrombin time as well as a moderate increase in the viscosity of the blood also attended the use of these substances.

TABLE III

EFFECT OF SMALL DOSES OF OXALIC ACID ON CLOTTING TIME AND OXALIC ACID CONTENT OF THE BLOOD

Clotting Time Seconds (Normal)	Oxalic Acid Mg./100 cc. (Normal)	Dose of Oxalic Acid, Mg.	Clotting Time Seconds	Oxalic Acid Mg./100 cc.
472	4.6	20	24	7.2
427	3.8	20	164	5.7
425	4.5	20	147	5.6
2,400	4.2	40	220	7.8
257	5.2	20	125	6.1
270	5.1	10	130	5.9
335	4.6	25	95	5.6
212	4.7	20	86	6.0
188	4.7	20	74	5.8

These plant extracts were clinically utilized in several thousand cases. Some of the results are depicted in Table IV. Subsequently it was found that in cases of prolonged coagulation time, in which there was a predisposition to hemorrhage, the oxalic acid level of the blood tended to be decreased (Table V). Moreover, in conditions in which the coagulation time was reduced and there was a predisposition to thrombus, the oxalic acid content tended to be increased (Table V). Schumann[9] stated that the plant extract was used with much success in the management of hemorrhage arising in obstetric and gynecologic cases; that in cases of uterine bleeding, either of the functional type or from fibroid tumor, the drug exerted an active influence; that in cases of fibroma the hemorrhage usually ceased within twenty minutes after intravenous injection and that, while it might recur, the quantity of blood lost was definitely much less. He observed "no toxic effects attending its usage." More recently patients with a wide variety of types of hemorrhage were treated by one of us (H. I. S.) with both the synthetic aqueous solutions of the dibasic acids and the oil solution of the ethyl esters, with rapid control of the bleeding. Instances of the use of both the synthetic and plant extracts preoperatively as a prophylactic measure, as well as postoperatively to control hemorrhage, were numerous.[2, 10, 11]

TABLE IV

RECENT CLINICAL RESULTS

Type of Bleeding	No. of Cases	Average Dose	Results
Epistaxis	43	2 cc. i.v.	Controlled in 5-60 min; average 15 min.
Exodontia	22	2 cc. i.v. 2 cc. i.m.*	Controlled in 10-30 min.
Jaundice	11	3 cc. i.v. 6 cc. i.m.*	Controlled in 10-45 min.
Hemophilia	15	3 cc. i.v.† 2-12 cc. i.m.*	Controlled in from 15 min. to 3 days.
Postpartum	20	3 cc. i.v. 2 cc. i.m.*	Controlled in 15-60 min.
Hematemesis	4	3 cc. i.v. 4 cc. i.m.*	Controlled in from 30 min. to 2 hrs.
Hemoptysis	32	3 cc. i.v. 6 cc. i.m.*	Controlled in 60% of cases within 2 hrs.
Purpura Thrombopenic	16	3 cc. i.v. 12 cc. i.m.*	Controlled in all but 1 case within 2 hrs.
Pretonsillectomy (prophylactically)	207	2 cc. i.m.	No postoperative ooze except in 8 cases.
Melena	25	3 cc. i.v. 6 cc. i.m.*	Controlled within 2 hrs.
Leukemia	3	3 cc i.v. 9 cc. i.m.*	2 failures; 1 controlled within 30 min.
Uterine bleeding	38	3 cc. i.v. 6 cc. i.m.*	Controlled within 1 hr. in most cases.
Bleeding from bladder	3	3 cc. i.v. 6 cc. i.m.*	Controlled with 30 to 60 min.
Chronis osteomyelitis	1	3 cc. i.v. 2 cc. i.m.	Controlled within 30 min.
Orbital hemorrhage	1	3 cc. i.v.	Controlled within 1 hr.
Postoperative (appendectomy)	2	3 cc. i.v. 9 cc. i.m.*†	Bleeding not controlled; reoperation; transfusions, etc.; complete control in 2 hrs. No recurrence.
Gasteroenterostomy	1	2 cc. i.v. 2 cc. i.m.	Controlled within 30 min.; no recurrence.
Partial mastectomy (prophylactically)	1	2 cc. i.v. 2 cc. i.m.	Clear field during operation; previously hemorrhaged from other breast.

The following abbreviations are used:—i.v., intravenously; i.m., intramuscularly.

*In divided doses of 2 cc.

†Entire course repeated on next day in 2 cases.

Maintenance treatment subject to cessation of hemorrhage often given.

The duration of the effect following the administration of this plant hemostatic varied with the degree of hemorrhage, the coagulation time, the amount of blood lost and the character of the hemorrage. In some cases of epistaxis the effect of 2 cc. lasted as long as three days. In a few cases, particularly when there was arterial bleeding, it was necessary to repeat this dose at hourly intervals to preserve the effect.

TABLE V

OXALIC ACID VALUES OF THE BLOOD IN HEMORRHAGIC STATES

	Mg. / 100 cc.
Normal	5.0 – 7.5 (6.5)
Familial hereditary epistaxis	2.9 – 5.4 (4.2)
Purpura haemorrhagica	2.6 – 5.2 (4.4)
Jaundice	3.4 – 6.0 (4.7)
Hemophilia	2.0 – 6.3 (4.6)
Uterine hemorrhage	5.1 – 6.4 (5.6)
Hemoptysis	4.6 – 5.8 (5.0)
Buerger's disease*	5.5. – 10.2 (8.7)

*Tendency to increased rate of coagulation.

Similarly, the doses of the hemostatic used varied with the duration and severity of the hemorrhage. In cases of severe bleeding, a regimen consisting of 3 cc. (six clotting units) given intravenously, followed immediately by 2 cc. (four clotting units) administered intramuscularly, with the intramuscular dose repeated at two hour intervals for three injections, usually sufficed. However, in some cases a dose of 2 cc. (four clotting units) given intramuscularly was repeated daily for several days after the successful treatment, to maintain the effect. More recently the initial dose of 3 cc. given intravenously and 2 cc. given intramuscularly was followed by 1 cc. of the ethyl ester in oil administered intramuscularly. This was repeated at intervals of one to three days, as indicated, and proved efficacious. Minor types of bleeding were usually controlled by 2 cc. (four clotting units) given intramuscularly. Others were treated with 3 cc. given intravenously followed by 2 cc. given intramuscularly, with good results.

TABLE VI

PLANT EXTRACT THERAPY FOR VARIOUS TYPES OF EPISTAXIS

Epistaxis	Duration and Severity of Hemorrhage	Treatment and Dosage	C.T. Before	C.T. After	Pct. Reduction	Supplemental Treatment	Prophylactic Treatment	C.T. Before	C.T. After	Pct. Reduction	Results	Comment
Chronic rheumatic heart disease	Profuse for 15 min.	2 cc. i.v.	4 min. 30 sec.	2 min. 20 sec.	48%	None	None	.....	.....	.....	Controlled in 12 min.	Previous hemorrhage required packing
Spontaneous	24 hrs.	2 cc. i.m.	.....	.....	.....	.....	.....	.....	.....	.....	Controlled in 15 min.; no immediate recurrence	Slight ooze following day; controlled in 15 min.; 2 cc. i.m.
Spontaneous	Very severe; almost exsanguinated	3 cc. i.v. 2 cc. i.m.	.....	.....	.....	.....	.....	.....	.....	.....	Controlled in 15 min.	.....
Hypertensive	Several hrs.	2 cc. i.m. 1 cc. i.m.	.....	.....	.....	.....	.....	.....	.....	.....	Controlled in 15 min.	No immediate recurrence
Spontaneous	Several hrs.	2 cc. i.m. 2 cc. i.v.	4 min. 50 sec.	2 min. 50 sec.	40%	None	2 cc. daily for 3 days	.....	.....	.....	Controlled in 9 min.	No recurrence

TABLE VI (Continued)

PLANT EXTRACT THERAPY FOR VARIOUS TYPES OF EPISTAXIS

Epistaxis	Duration and Severity of Hemorrhage	Treatment and Dosage	C.T. Before	C.T. After	Pct. Reduction	Supplemental Treatment	Prophylactic Treatment	C.T. Before	C.T. After	Pct. Reduction	Results	Comment
Traumatic	Severe; 1 and ½ hr.	2 cc. i.v.	.....	.....	.....	.....	.....	.....	.....	.....	Controlled in 28 min.	.....
Spontaneous	Severe; 3 days	3 cc. i.v. 2 cc. i.m.	.....	.....	.....	.....	.....	.....	.....	.....	Controlled within 30 min.	No recurrence
Hereditary familial epistaxis	7-9 daily, last 15 min.	.....	7 min. 51 sec.	2 min. 27 sec.	69%	None	2 cc. 3X a week	.....	.....	.....	No. and severity markedly reduced	.....
Spontaneous	Severe; several hrs.	3 cc. i.v. 2 cc. i.m.	6 min. 15 sec.	4 min.	36%	None	.....	.....	.....	.....	Within 30 min.	No recurrence
Polycythaemia vera	Severe; 4 hrs.	2 cc. i.v. 2 cc. i.m.	10 min.	5 min. 25 sec.	49%	None	2 cc. i.m. daily for 3 days	5 min. 25 sec.	2 min. 30 sec.	54%	Controlled in 30 min.	No recurrence
Chronic osteomyelitis	Severe; several hrs.	3 cc. i.v. 2 cc. i.m.	24 min.	7 min.	71%	None	.....	.....	.....	.....	Controlled in 30 min.	Hemarthrosis 30 days later

Prophylactically, 2 cc. (four clotting units) was given intramuscularly one-half hour before operation, followed by 2 cc. administered intravenously and 2 cc. administered intramuscularly immediately after operation.

The dosage may be varied according to the discretion of the physician. In one case of hemophilia with severe hematuria, it was necessary to give 3 cc. of the drug intravenously and 2 cc. intramuscularly, followed by 2 cc. given intramuscularly at two hour intervals for three injections and 3 cc. given intravenously and 2 cc. intramuscularly daily for two weeks to maintain control of the hemorrhage.

TABLE VII

PROPHYLACTIC EMPLOYMENT OF THE PLANT EXTRACT IN TONSILLECTOMIES

	Control	Test
Total number of cases	125	157
Number of cases of primary bleeding	28	7†
Per cent of primary bleeding	22.4%	4.5%
Number of cases of secondary bleeding	2	1‡
Per cent of secondary bleeding	1.6%	0.7%
Total per cent of bleeding	24 %	5.2%
Number of local tonsillectomies	42	42
Number of cases of bleeding	7	2*
Per cent bleeding following local tonsillectomy	16.7%	4.8%
Number of general tonsillectomies	83	115
Number of cases of bleeding	23	6
Per cent bleeding following general tonsillectomy	27.7%	5.2%
Coagulation time before extract		1 min. 50 sec. to 10 min.
Coagulation time after extract		1 min. 20 sec. to 4 min. 50 sec.
Av. reduction in coagulation time after 2 cc. extract		41.6% within 30 min.

For the controls, therapy consisted of pressure, tannic acid, ice cap, morphine, calcium gluconate and snake venom.

Test therapy consisted of plant extract, 2 cc. (4 clotting units).

†Required additional 2 cc. (4 clotting units).

‡Pressure and plant extract (8 cc. [16 clotting units]).

*Hypertension a contributory factor.

A sufficiently large series of cases of hemorrhage encountered in the practice of otorhinolaryngology has been observed to justify using these hemostatic preparations routinely. A group of patients with epistaxis due to various causes were treated with both the plant and the synthetic extracts. Some of the results are depicted in Table VI. The average reduction in coagulation time within 30 minutes after the administration of the drug was 52 per cent. In one case of chronic osteomyelitis, in which there was considerable erosion of the septum, the initial clotting time was 24 minutes, with a resultant decrease of 71 per cent within 30 minutes of administration of the drug. Several patients with recurrent episodes of bleeding were treated prophylactically, with elimination of subsequent hemorrhage or a marked reduction in the amount and duration of flow. Another group of 157 patients scheduled for tonsillectomy (Table VII) were given 2 cc. (four clotting units) of this plant hemostatic intramuscularly one-half hour to one hour before operation. Of the patients so treated, 7 (4.5 per cent) had primary bleeding, which was controlled by an additional postoperative dose of 2 cc. In 1 case (0.7 per cent) secondary bleeding occurred and could not be controlled until an additional 8 cc. of hemostatic was administered and pressure was applied. There was an average reduction of 41.6 per cent in the coagulation time for the treated patients. This appears to be significant when compared with the condition of a group of 125 control patients who received no prophylactic treatment, of whom there were 28 (22.4 per cent) who had primary bleeding and 2 (1.6 per cent) with secondary ooze. In this series therapy consisted of applying pressure and an ice cap, tannic acid locally, or the administration of morphine sulfate, calcium gluconate or snake venom, or a combination of these measures. It may be significant that the number of instances of bleeding following general anesthesia was approximately three times as great as that encountered with the use of local anesthesia, although in most of the former the patients were children.

SUMMARY

A hemostatic prepared by the extraction of certain plants is reported.

This hemostatic can be effectively administered intravenously or intramuscularly without untoward effects.

A clotting unit has been devised which measures the hemostatic potency of the extract.

The active principle of this extract and some similar ones was found to be oxalic acid and certain related dicarboxylic acids. Sev-

eral thousand patients with various types of hemorrhage were treated, with a high incidence of gratifying results.

Solutions of various combinations of these chemically pure acids manifested various degrees of hemostatic activity.

A method for the determination of the oxalic acid content of the blood is described. Normal values for human beings by this method were 5 to 7.5 mg per 100 cubic centimeters.

In cases of epistaxis, significant results were obtained in the control and prevention of bleeding.

The prophylactic use of this hemostatic in tonsillectomy resulted in an important reduction in the incidence of primary and secondary bleeding.

The hemostatic successfully controlled the hemorrhage incident to tonsillectomy.

LAWNTON AND OAKLANE AVENUES.

REFERENCES

1. Steinberg, Arthur: A New Blood Coagulant: Its Use in Treatment of Hemorrhage. Read before the Medical Society of Roselle and Roselle Park, New Jersey, December 6, 1938.

2. Milliken, L. F.: The Use of a New Blood Coagulant in Transurethral Prostatic Resection. J. Urol., 42:75 (July), 1939.

3. Steinberg, Arthur, and Brown, W. R.: A New Concept Regarding the Mechanism of Clotting and the Control of Hemorrhage. Am. J. Physiol., 126:638 (July), 1939.

4. Suzuki, S.: On the Oxalic Acid in the Blood. Jap. J. M. Sc. Tr., II, Biochem., 2:291 (June), 1934.

5. Ersner, M. S., Myers, D., and Ersner, W.: Clinical and Experimental Study of the Action of Saliva on Blood Coagulation and Wound Healing in Surgery of the Oral Cavity and Throat. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 43:114 (March), 1934.

6. Hammersten: Zur Lehre von der Faserstofsgerinnung. Arch. f.d.ges. Physiol., 14:211, 1877.

7. Horne, R. M.: The Action of Strontium, Barium, and Calcium Salts in Preventing Coagulation of Blood. J. Physiol., 19:356, 1896.

8. Pickering, J. W., and Hewitt, J. A.: The Action of Oxalates and of Citrates on Circulating Blood. J. Physiol., 59:455, 1924.

9. Schumann, E. A.: Newer Concepts of Blood Coagulation and Control of Hemorrhage. Am. J. Obst. & Gynec., 38:1002 (Dec.), 1939.

10. Jackson, A. S.: Editor's Comments—Bleeding Ulcer. Jackson Clin. Bull., Madison, Wis., 2:3 (Jan.), 1940.

11. Campbell, K. N.: Personal communication to the authors.

METHOD FOR DETERMINATION OF OXALIC ACID IN THE BLOOD*

1. To 2 cc. of serum (unhemolyzed) in a test tube add 4 cc. of 10 per cent trichloroacetic acid.
2. Stir and centrifuge rapidly for five minutes.
3. Pour off and retain the supernatant filtrate.
4. Put 3 cc. of the trichloroacetic acid filtrate into a 15 cc. centrifuge tube (pyrex) with a constricted tip.
5. Add 1 cc. of 0.1 per cent cerium chloride.
6. Adjust the pH with 5 per cent sodium hydroxide drop by drop until just alkaline to congo red, and stir vigorously after the addition of each drop until the appearance of a fine precipitate.
7. Allow to stand for thirty minutes.
8. Centrifuge rapidly for five minutes.
9. Pour off and discard the supernatant fluid.
10. Allow the inverted tube to drain on filter paper for five minutes.
11. Wash the precipitate twice with 2 cc. of distilled water and discard the washings.
12. Dissolve the precipitate in 3 cc. of 1.5 normal sulfuric acid.
13. Place the tube in a boiling water bath for one minute to facilitate solution.
14. To a similar 15 cc. centrifuge tube add 1 cc. of 0.01 per cent oxalic acid and 3 cc. of 1.5 normal sulfuric acid (standard).
15. To standard and unknown add 1 cc. of 0.05 per cent normal potassium permanganate.
16. Stir vigorously and titrate at room temperature against 0.05 per cent normal sodium oxalate until the pink color just disappears.

Calculation:

$$\frac{\text{(Titration of unknown subtracted from 1)}}{\text{(Titration of standard subtracted from 1)}} \times 100 = \text{mg. oxalic acid per 100 cc. of blood}$$

*A modification of Suzuki's method.[4]

LXXXIV

THE SURGICAL ESTABLISHMENT OF NASAL VENTILATION

SIDNEY N. PARKINSON, M.D.

OAKLAND, CAL.

Nasal physiology is concerned with respiration and olfaction, with warming and humidifying inspired air, controlling infection, and modifying the voice. All of these functions depend upon adequate nasal ventilation permitting normal passage of the tidal air of respiration. Sufficiency of ventilation, therefore, is the structural basis of nasal function.

Nasal obstruction and the inevitable impairment of function deriving from it constitute a common and important problem. The symptoms are distressing, and the pathologic by-products are not entirely reparable. Supplementary mouth breathing, ear blockage, prolongation of colds, and reduction of taste and smell are common local complaints. Fatigability and malaise are not unusual general symptoms. Thickening of nasal and sinal membranes predisposes to sinusitis. Tympanic changes leading to restricted mobility of the conduction apparatus contribute to reduction of hearing.

The restoration of function requires the re-establishment of breathing space. In some cases this is accomplished by nasal or sinal treatment or by surgery directed primarily toward relief from sinusitis. In others relief is secured through control of the existing allergy by elimination or desensitization. In still others help comes through regulation of metabolism. Present interest, however, lies in the cases which under such regimes receive insufficient relief or none. Among these are many who quite properly require surgery.

Too often intranasal surgery has been disappointing as a means of restoring breathing space. Generally speaking, the reason seems to lie not in faulty surgical technique but in over-standardization of approach, or more specifically, in over-dependence on some one procedure. The surgery has not been designed specifically to meet the particular pathological condition. A variety of pathologic entities contribute to nasal obstruction. Their correction requires a variety of procedures. Only thus may one correlate surgery to pathology.

Fig. 1. Normal nasal ventilation. Mrs. N., age 32. Colds infrequent and mild. No hay fever. Pharynx, ears, and sinuses normal. Tonsillar pillars show no redness. Septum straight. Nosal mucosa pink-red, clean, not swollen.

Fig. 2. Nasal obstruction. Mrs. K., age 46. Complaint of difficulty in nasal breathing for many years. Obstruction is seen to be swollen mucous membrane, particularly of inferior turbinates.

The following case report illustrates a situation often encountered:

Mrs. K. complained of difficulty in nasal breathing since girlhood, worse on the right side, and of frequent ear blockage for ten years or more, worse on the right side. The history includes many local and general examinations and treatments. A submucous resection had failed to improve the breathing.

Examination revealed a nasal mucous membrane, dull red, clean and much congested. Hyperplasia was evident along lower border and at posterior tip of both inferior turbinates. No discharge was seen and none complained of except during colds. The septum was straight. The maxillary sinuses were reported "slightly cloudy."

That the long-standing nasal blockage was due to swollen mucous membrane was obvious. Shrinkage by means of a vasoconstrictor provided comfortable ventilation for an hour or so, after which the membrane resumed its persistent congestion.

The four main causes of nasal obstruction of the chronic type, each a separate entity, are:

1. Deviated septum.
2. Persistent mucosal congestion.
3. Persistent mucosal edema.
4. Mucosal hyperplasia.

Another cause, alar collapse, is both closely related and pertinent to the subject. The pathology, diagnosis, and treatment of this condition have been recently discussed by Lillie and Simonton.[1] Polyposis, adhesions, and choanal atresia are outside the scope of this presentation.

The etiology of deviation of the septum need not concern us here. Persistent mucosal congestion can be caused by irritation as from infection, or it can be metabolic as from thyroid deficiency. Persistent edema is apt to be allergic in origin. Hyperplasia results from persistent congestion, persistent edema, or both.

Congestion, edema, and hyperplasia, while affecting all nasal membrane, involve most extensively the erectile tissue of the inferior turbinates. At both nostril and choana the turbinate stands directly in the path of the air stream. This serves a physiologic purpose, but it serves likewise to accentuate the obstructive importance of turbinal abnormalities.

To correct the results of nasal obstruction five surgical procedures deserve consideration. All are based on established surgical

Fig. 3. Same case as "2". Shrinkage of mucosa with a vasoconstrictor has temporarily restored ventilation. This patient had had a submucous resection two years previously, with no noticeable effect on breathing space.

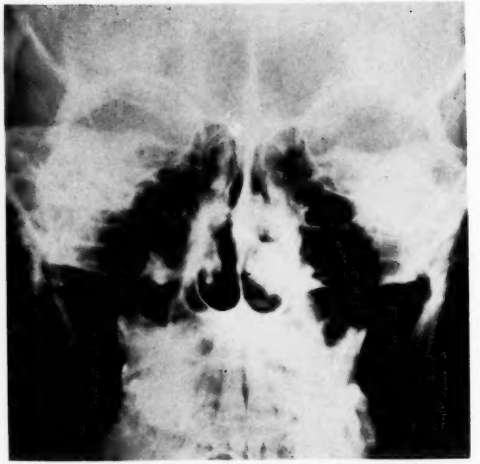


Fig. 4. Nasal obstruction. Dr. M., age 34. Difficulty in nasal breathing since boyhood. Had had a submucous resection few years previously with no noticeable improvement. Obstruction is seen to be swollen mucous membrane, particularly of inferior turbinates.

principles. One of them, intranasal antrostomy, includes a new technical detail. The aim of all is to achieve ventilation by restoring intranasal architecture to approximately the normal pattern. These procedures are:

1. Submucous resection.
2. Resetting of inferior turbinate.
3. Reduction of inferior turbinal bulk.
4. Antrostomy.
5. Reduction of middle turbinal bulk.

By co-ordination of these procedures one may usually solve the ventilation problems created by intranasal pathology.

Submucous resection if deemed necessary should be performed with particular attention to areas most important in nasal respiration. These are the junction of the septum and floor anteriorly, the post-columellar area of cartilage, and the cartilage adjoining the nasal bridge. Obstruction in these areas must be sufficiently removed.

Resetting of one or both inferior turbinates usually is advantageous, often imperative. By this is meant lateral fracture or displacement of the turbinate so as to increase the space between it and the septum. For example, in a low septal deviation the turbinate on the concave side is apt to be large, medially placed, and persistently swollen. Septal straightening without attention to the turbinate leaves the passage on that side more blocked than before. Pressing the turbinate laterally increases breathing space and tends to establish symmetry and equality between the two sides.

Reduction of inferior turbinal bulk.—It may be advisable to reduce the size of one or both inferior turbinates. The procedure depends upon the pathology whether it be hyperplasia, congestion or edema. Hyperplasia is most common along the lower border and at the posterior tip. If the erectile tissue is thoroughly shrunken with a vasoconstrictor, hyperplasia becomes evident as a dependent fold of tissue movable with a probe. It is redundant tissue, the result of disease, and serves no purpose. It may well be trimmed off.

Undue swelling of erectile tissue, whether from congestion or edema, can be corrected to a considerable extent. A sharp instrument is applied here and there to the turbinal surface, preferably

penetrating through the erectile tissue to the periosteum. Healing leads to increase in connective tissue which limits the extent of subsequent swelling. This method is preferable to escharosis because of the greater control of turbinal reduction and the reduced reaction and discomfort.

Antrostomy.—Subturbinal antral windows heretofore have been made for the purpose of reducing infection within the sinus. Another purpose is now evident. Resetting the inferior turbinate to a more lateral position now places it where its lateral surface adjoins a more or less free space. During subsequent congestion or edema this surface swells partly into the window. Otherwise impingement on the nasal wall often causes it to return to a more medial position. The procedure has proved to be decidedly useful and in many instances has appeared to be essential to success. Undoubtedly also, a more healthy sinus, resulting from the increased sinal ventilation, contributes in many instances to less turbinal congestion.

The middle turbinate seldom is important in the problem of nasal obstruction, although it not infrequently is important in sinal ventilation. If needed, however, corrective measures such as compression and partial submucous resection are well known.

OPERATIVE PROCEDURE

The usual precautions as to normal bleeding and clotting time and freedom from acute infection are followed. The surgery may be done under local anesthesia, preferably from novocain injected intramucosally and submucosally. Postoperative packing is inadvisable. Packing serves no useful purpose, and its avoidance relieves the patient of the chief source of postoperative discomfort. Bleeding is seldom profuse and almost always stops within a reasonable time. In the occasional case in which it does not stop spontaneously, control by choanal and nostril tampons is resorted to. Their removal does not disturb the operative field. No special problems have been encountered.

RESULTS

By correlating surgery to pathology, satisfactory nasal ventilation is achieved with gratifying frequency. The mucous membrane becomes cleaner, while its brighter redness indicates a more active blood supply. After five years' experience I can say that ventilation surgery has become a most satisfactory procedure. Certainly there

are few more satisfied patients than those to whom good nasal breathing has been restored.

SUMMARY

Inadequate nasal ventilation is a common and important problem. It is due to a variety of pathologic entities, the most usual being persistent congestion, persistent edema, deviated septum, and mucosal hyperplasia. Many cases are relieved by allergic or endocrine control, or by nasal or sinal treatment. The remaining cases usually can be relieved by intranasal surgery following sound and approved principles. As the causes of chronic obstruction vary, surgical measures must vary accordingly. Besides submucous resection, measures are outlined for resetting the inferior turbinate, reducing its erectile capacity, and removing portions that are hyperplastic. Intranasal antral windows are useful as additional space for resetting the inferior turbinate. This is recommended as a valuable addition to ventilation surgery. Improved ventilation brings improvement in nasal physiology.

1904 Franklin Street.

REFERENCE

1. Lillie, Harold I., and Simonton, Kinsley M.: Nasal Obstruction Caused by Collapse of the Nasal Alae. Annals of Otology, Rhinology and Laryngology, 48:600 (Sept.), 1939.

The

ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

ANNOUNCES ITS

JUBILEE VOLUME

1941

IN 1941 the ANNALS will have been in continuous existence for fifty years. The four issues comprising

VOLUME FIFTY

will constitute a Jubilee Volume in which will appear fifty articles written by invitation and contributed especially for this work by the most eminent contemporary American laryngologists.

FIFTY years of unswerving devotion to the ideals of laryngology and uninterrupted service to its devotees throughout the world will soon be the enviable record of the ANNALS. The four issues to appear in 1941 will constitute VOLUME FIFTY.

TO COMMEMORATE this inspiring occasion the four issues will be known as the Jubilee Volume and will contain, in part, fifty articles written specially for it by leading American laryngologists. This volume will represent a cross-section of contemporary thought and practice in the specialty, and will be in effect a reference book for years to come.

TO THIS end the Annals Publishing Company will arrange, for the convenience of the subscribers who wish it, to bind the four issues, at the end of the year, in a distinctive binding at a minimal cost.

IT HAS always been the purpose of the editors of the ANNALS to print first-hand reports of the foremost advances in otolaryngology and its borderline sciences with completeness and authenticity, avoiding wherever possible the untried, the purely hypothetical, and the repetitious. From this there will be no deviation in the future. Illustrations, format, and typography which have always been of a high standard of excellence will continue to keep pace with the best practice of the times.

ANNALS PUBLISHING COMPANY

Editorial Office: 3720 WASHINGTON BOULEVARD

Business Office: 7200 WYDOWN BOULEVARD

ST. LOUIS

Clinical Notes

LXXXV

AIR RIFLE BUCK SHOT IN LEFT POSTERIOR ETHMOIDS: OPERATIVE REMOVAL*

M. Gerard Golden, M.D.

Brooklyn, New York

The following case is of unusual interest because of its circumstances and the absence of a similar operative removal in the literature.

P. S., a 13-year-old white schoolboy, on the advice of his private physican came to the Eye Clinic of the Outpatient Department of the Kings County Hospital, on January 22, 1940.

Four days before admission he was accidentally shot in the left eye with a single air rifle buck shot by a playmate, who was at a distance of about ten feet from the patient when the rifle discharged. They were playing Indians in the cellar, and the boy picked up the rifle unaware of the fact that it was loaded.

Physical examination was entirely negative except for the local condition, which exhibited some redness of the scleral vessels of the left eye and a punctate wound through the sclera on a level with the canthus, midway between the inner canthus and the medial margin of the iris. The structures in the left side of the nose showed no evidence of trauma, but there was a marked congestion of the nasal mucosa on that side. Emergency X-ray studies were made and the patient was advised to enter the hospital on the eye service.

The X-ray findings were reported as follows: "Evidence of spherical foreign body in the region of the left orbit, localized in the left posterior ethmoids."

Examination of the X-ray films showed the shot to be less than an eighth of an inch under the roof of the left posterior ethmoid cell and a fourth of an inch forward from the anterior wall of the sphenoid sinus, insofar as one may approximate these distances from the

*From the Department of Otolaryngology of the Kings County Hospital.

usual postero-anterior and lateral exposures of the sinuses to the X-rays.

The patient was transferred to the Ear, Nose and Throat Service with the following note: "X-rays definitely show shot imbedded in left posterior ethmoids. Conjunctival point of entry just below internal rectus muscle insertion." An emergency medical consultation was held. There was no contra-indication to surgery from the medical standpoint. The blood pressure was 128/60.

The usual way to remove a bullet or rifle shot is by probing through the portal of entry, if the bullet has not penetrated too far from the site of entry. In this case the usual approach, namely, the site of entry, was immediately abandoned, being the eye, and the X-ray showing the shot in the ethmoids. The other two methods of approach to this problem could be an intranasal ethmoidectomy or an external operation. The author chose the intranasal approach, and a left intranasal ethmoidectomy was accordingly done.

Under ether anesthesia a postnasal plug was inserted, and the left middle turbinate was infracted medially against the septum.

With a small biting forceps the posterior ethmoid cells were uncapped, presenting the foreign body, a small brass-coated air rifle buck shot, to view. This was removed with cupped nasal grasping forceps. A minimal amount of bleeding was encountered. This was controlled with a few cotton tampons soaked in adrenalin. No nasal packing was used.

At 9:00 p. m. the same night the patient received 1500 units of tetanus antitoxin intramuscularly. The delay in giving this was caused by the necessity of an inquiry as to whether or not it had been given previously.

There was no bleeding. There was a slight amount of blood-tinged mucus in the left side of the nose. The patient made an uneventful recovery. The temperature, pulse and respiration were within normal limits.

Urinalysis and the blood Wassermann test were negative. Urea, creatinine and sugar were present in the blood in normal concentrations. The patient was discharged on January 27, 1940, and instructed to report to the Ear, Nose and Throat Dispensary.

On subsequent postoperative visits to the dispensary, his left nasal meatus revealed a minimal inflammation. Healing was complete in 11 days. He was referred to the Eye Dispensary for further

follow-up. On February 5, 1940, the patient showed a slight pinhead-sized stellate scar at the point of entry in the conjunctiva. Careful fundus examination at the extreme nasal periphery disclosed extensive choroidal tears with beginning retinitis proliferans. Hemorrhages were being absorbed. On February 26, 1940, the findings were as above. The vision in the left eye was 20/20.

COMMENT

A review of the literature reveals a surprising paucity of reports of foreign bodies in the paranasal sinuses and no similar operative case. Salinger[1] quotes Okonogi[2] who reports a case of a one-year-old child shot with an air rifle. The wound was in the region of the right side of the nasal root. X-ray plates showed the bullet in the ethmoid sinus. Nasal examination revealed a bloody area in the upper portion of the middle turbinate. Probing was ineffectual. Three days later the foreign body was expelled in the feces.

Salinger[3] further quotes Leite[4] who reported a case involving the sphenoid sinus. "A revolver bullet entered the outer part of the eyelid and penetrated into the sphenoid sinus directly under the sella turcica. The bulb was destroyed, and there was a temporary paralysis of the legs, which was attributed to hemorrhage in the medullary sheath. The foreign body had been present for five years at the time of writing, without any further symptoms. The author was able to find only eight similar cases reported in the literature up to 1935."

Two cases involving the maxillary antrum have been reported. Smyth[5] mentions d'Onofrio[6] who reported a case of removal of a small grenade projectile which had been lodged in the antrum for sixteen years.

The other case is quoted by Salinger[7] who mentions the case of Hirayama[8]. The latter reports a man of 38 who while operating a wood cutting machine was struck in the cheek with a flying splinter which he removed from the soft tissues himself. Three days later the cheek became swollen, painful and tender, and a discharge appeared at the point of entry. Examination showed a fistula at the upper end of the nasolabial fold, which was lined with granulation tissue, bled freely and admitted a probe to the depth of 5.5 cm. A Caldwell-Luc operation revealed a defect in the anterior wall of the sinus, covered partly by a fragment of bone, suppuration within the cavity and a small splinter of wood embedded in the granulation tissue.

CONCLUSION:

1. A case is reported of an air rifle buck shot in the left posterior ethmoid sinus. It was removed by intranasal ethmoidectomy. The results were satisfactory.

2. Although the left eyeball showed definite signs of injury, the patient had perfect vision in that eye one month after the injury.

3. A survey of the literature shows very few reports of foreign bodies in the paranasal sinuses and none of a foreign body in the ethmoid sinus removed by operative intervention.

1 NEVINS STREET.

BIBLIOGRAPHY

1. Salinger, S.: Progress in Otolaryngology—The Paranasal Sinuses. Arch. Otolaryng., 24:384 (Sept.), 1936.

2. Okonogi, S.: Ein Spont. ausgestossener Fremdkórper der Siebbeinzellen, Otologia (Fukuska) 8:807, 1935; abstr. Zentralbl. f. Hals-, Nasen- u. Ohrenh., 25:632, 1935.

3. Salinger, S.: Progress in Otolaryngology—The Paranasal Sinuses. Arch. Otolaryng., 26:374 (Sept.), 1937.

4. Leite, A. O.: Ein seltener Fall von Fremdkörper (Revolverkugel) im limken Sinus Sphenoidalis, Arq. clin. oftal. e otol., 2:108, 1935.

5. Smyth, D. C.: Progress in Otolaryngology—The Paranasal Cavities. Arch. Otolaryng., 18:219 (Aug.), 1933.

6. d'Onofrio, F.: Lodging of a Small Grenade Projectile in the Maxillary Sinus for 16 Years: Removal; Report of a Case. Arch. ital. di. otol., 43:65 (Feb.), 1932.

7. Salinger, S.: Progress in Otolaryngology—The Paranasal Sinuses. Arch. Otolaryng., 30: 668 (Oct.), 1939.

8. Hirayama, S.: Fremdkörper in der Kieferhöhle infolge Trauma. Oto-rhino-laryng., 11:219 (March), 1938.

LXXXVI

LAVAGE OF THE ANTRUM WITH A BLUNT INSTRUMENT THROUGH THE MIDDLE MEATUS

(A Series of 2,580 Cases)

W. C. Bowers, M.D.

New York

Material for this study was provided by the office records of my private practice. These records show that since the year 1925 the only cases irrigated by puncture through the inferior meatus were those in which the middle meatus was dangerous because of anatomical formation; these amounted to five or six in all. Notations were found on 2,580 primary irrigations with a blunt cannula through the middle meatus; 1,223 on size of normal or accessory openings; and 757 on position of openings.

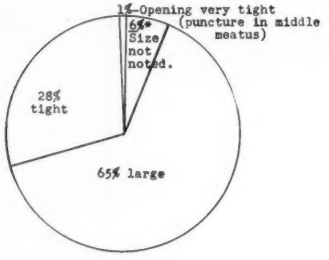


Fig. 1.

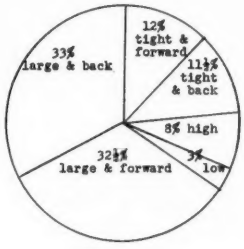


Fig. 2.

Fig. 1. Size of antral openings in 1,223 cases. *This 6% is presumably good size as originally no note was made unless opening was unusual.

Fig. 2. Position of antral openings in 757 cases.

FACTS ABOUT IRRIGATING THE ANTRA THROUGH THE MIDDLE MEATUS

1. Blunt irrigation is as thorough as puncture.

2. When once the technique is developed, the operator and the patient will not be likely to want the puncture.

3. There is much less trauma and much less likelihood of complications.

4. It can be used on people who would not permit puncture; therefore, exploratory washing can be more readily performed and more disease of the antra discovered.

5. Repeated washings are much better tolerated.

6. Danger of hemorrhage is much reduced.

7. It can be used on children as young as four years of age.

8. There is no shock as there is very little noise or pain.

9. A blunt-ended instrument permits of better tactile sense and slides into an opening much more easily than an open end catheter which has a tendency not to slide so readily.

10. Beware of the deep middle meatus as this often indicates a small antrum and close proximity of the orbit.

11. Of 2,580 primary irrigations of the antra by this means not over six mishaps have occurred; these consisted of penetration and introduction of fluid into the orbit (five cases) and the cheek (one case), which abated in a few hours without any infection.

17 E. 61ST ST.

LXXXVII

CARCINOMA, PRIMARY IN THE EUSTACHIAN TUBE: REPORT OF TWO CASES, ONE WITH CURE

PHIL W. SHUMAKER, M.D.

LOS ANGELES

Examination of the American literature fails to reveal a record of the diagnosis during life, and of the treatment, of malignant tumors involving the eustachian tube other than secondarily from the nasopharynx. Ewing states that he has never seen a proven case of primary eustachian tube carcinoma and knows of none in the literature. New's excellent paper on nasopharyngeal tumors makes no mention of such an entity. It is true that this year Brownell has reported two cases of carcinoma of the eustachian tube, both of which, however, were diagnosed at autopsy. The following two cases, therefore, represent the first recorded in the American, and apparently in the English, literature of the diagnosis of primary eustachian tube carcinoma while the patients were alive, and of the treatment with cure in one case and with the outcome unknown in the second because it was impossible to follow the patient. Both of these cases were presented before the Research Study Club of Los Angeles in 1933. It is felt that the definite clinical picture and pathological findings, practically duplicated in the two instances, may prove of service in aiding earlier diagnosis in similar cases.

CASE 1.—Mrs. S., aged 54, a white Austrian woman, was seen by me only twice, in the spring of 1931, at the Otologic Clinic of Prof. Erich Ruttin in the Rudolfspital in Vienna. The patient complained of deafness on the left side for the previous two months. Examination on the right revealed normal tympanic membrane and hearing, while the left side showed the typical yellowish tympanic membrane through which could be seen a fluid level. The records showed that this patient had received bi-weekly inflations for the previous six weeks, and she stated she could see no benefit from the treatments. The records also showed the nasopharyngeal examination to have been negative on the first visit, tubal orifices being specifically mentioned as normal. When I examined the nasopharynx with a mirror, I found a rounded, reddish, cauliflower-like growth issuing from the left eustachian orifice. The tumor at this time

Fig. 1. Schematic (tumor in the tube mouth).

had an approximate diameter of 12 millimeters and was movable as if it were attached by a pedicle within the tube. A specimen was taken, which proved to be a squamous epithelioma of the highest malignancy. (Note: The appearance of the tumor and the biopsy sections are practically identical with those made of and shown with Case 2.) On her next visit she was ordered to the government radium institute for therapy, and I regret to report that I neither saw nor heard of this patient again. It is possible that she was saved by the radium, but obviously her chances would have been greatly enhanced had a weekly examination of the nasopharynx been made and the tumor discovered when it first appeared in the eustachian orifice.

CASE 2.—Mr. J. W. S., aged 60, white, an apparently healthy American, was first seen at the Graves Memorial Clinic, Los Angeles, on March 15, 1932. He complained of deafness and a full feeling in the left ear, present continuously for the previous six weeks despite weekly inflations of the eustachian tube by a private physician. His claim of total deafness on the right since childhood was verified.

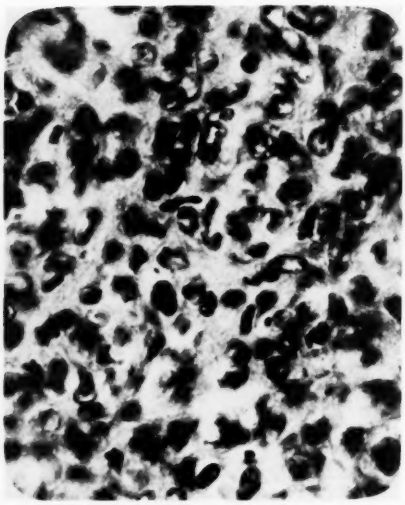


Fig. 2. Photomicrograph of squamous cell epithelioma, Grade IV. (Magnified X 400.)

The tympanic membrane was normal. Hearing on the left had been markedly impaired for many years, but had been sufficient for daily work and conversation. When seen, he could not hear a watch tick at the left concha, but could hear loud conversation. The left tympanic membrane was typically yellow, and through it was seen fluid at a high level. Mirror examination of the nasopharynx disclosed normal findings, including both eustachian orifices. Catheter inflation was accomplished, but not through what could be termed a clear tube. Bubbles appeared in the fluid, but it remained, and the hearing was not improved.

Nasal instillations of ephedrine and bi-weekly catheter inflations for the following two weeks altered neither the signs nor symptoms. Paracentesis was considered but deferred.

In the third week of my treatment (ninth week from onset) the tube seemed a trifle more obstructed. When this was again noted in the fourth week, a growing conviction, based on the experience with the first case reported, prompted the probable diagnosis of primary carcinoma of the left eustachian tube. At this visit I called two other otolaryngologists in consultation, and asked them

to observe him with me, once weekly. Careful inspection of the left tympanic membrane and eustachian orifice was made weekly by each examiner in the presence of the others.

On the third such weekly examination (the seventh week of the author's examinations, and the thirteenth week since the onset of symptoms) I discovered a very minute, reddish, cauliflower-like growth within the mouth of the left eustachian tube. One consultant was able to verify this finding that day, the other agreed the following week that now he could see the new growth also.

Three weeks after it became visible, the tumor had bulged out of the opening and was sufficiently large to permit biopsy (Fig. 1). Under local anesthesia, through a Yankauer speculum, a specimen was taken without pain or hemorrhage. This was on May 28, 1932, just sixteen weeks after onset of symptoms, during which time the patient's general health, appearance, appetite, weight, et cetera, had remained normal, and he had experienced no pain in the ear or nasopharynx, and had had no headaches, bleeding, or unusual symptoms other than the original complaint.

The biopsy report was Grade IV squamous epithelioma (Fig. 2). Therapy was begun June 9, 1932, when, under local anesthesia, a gold radon seed containing 2.1 millicuries of radium emanation was buried directly in the center of the tumor to a depth of 7 or 8 mm. This was done in the presence of several members of the Soiland Clinic, who then gave the patient 300 units of deep x-ray over the left side of the neck. Although no left cervical glands were ever palpable, x-ray therapy was repeated three times, at weekly intervals, as a prophylactic measure. No other therapy to the eustachian orifice or neck was used then or since.

Seven days later the tumor was pale. The hearing was slightly improved. There had been a hemorrhage into the left middle ear; paracentesis released one large drop of dark blood. Sixteen days after the radon seed was inserted, the tumor appeared half its former size. It and a surrounding area of 15 mm. looked very pale with no sloughing. The paracentesis wound was healed, and the membrane looked nearly normal. The patient heard a watch tick at the concha for the first time in five months. On the 33rd day he reported by phone that three small pieces of white "gristle" came out while he was gargling. On the 51st day after the radon application, the left eustachian orifice and membrane both appeared normal in all respects, with no evidence of tumor. The patient heard a watch tick at two inches.

On August 15, 1932 (65 days after treatment) and thereafter, the hearing of the watch tick remained constant at 12 to 14 inches, which the patient stated was as well as he had heard for many years. Low conversation was heard well. The appearance of the eustachian orifice and the tympanic membrane remained normal at every examination thereafter, the patient being seen at intervals no longer than monthly for three years. Every complaint throughout this period was promptly checked by physical and laboratory methods, nothing of note being found until early in 1935, when he began to complain of epigastric distress on exertion. A most thorough examination showed normal blood, urine and stool, negative lungs and abdomen, and normal gastrointestinal examinations. Heart examination and electrocardiogram indicated a coronary disease, and treatment was instituted.

He improved for some months, but on July 21, 1935, suffered a typical coronary accident from which he died two weeks later, on August 4th. Notification of his death was not received until 36 hours later, hence only a limited autopsy could be obtained. The region of the left eustachian tube was studied, and all structures, including the tube and its mouth, were found to be macro- and microscopically free of any tumor-like cells.

In view of the thorough and repeated antemortem examinations, the postmortem findings, the absolute lack of anything suggestive of recurrence, and the typical coronary death in a known coronary disease, it is safe to consider this a definite cure of over three years of an early carcinoma, primary in the left eustachian tube.

COMMENT

All textbooks give the early signs of nasopharyngeal tumors as (1) pain, (2) obstruction of nasal breathing, (3) obstruction of the eustachian tubes, and (4) postnasal bleeding. A primary tumor arising within one eustachian tube produces none of these signs except blocking of the one tube involved. If we are to wait until the textbook signs appear, from secondary invasion of the nasopharynx, the possibility of cure will usually be beyond our present limitations.

Therefore, in cases of unilateral secretory catarrhal otitis which do not respond to the usual methods of treatment after two to four weeks, a tumor of the eustachian tube should be suspected and perhaps may be diagnosed weeks before any visible growth appears, as was done in the second case herewith reported. At any rate, all

cases of catarrhal otitis, whether dry or secretory, should have a thorough examination of the nasopharynx, and if they do not respond to treatment as expected, should have a weekly examination of the eustachian orifices until they either improve or the cause for their failure to do so is known. The diagnosis of such an early case offers hope in its treatment.

It is worth mentioning that neither of the patients reported had ever had a eustachian bougie used.

CONCLUSIONS

1. Primary tumors originating within the eustachian tube, while rare, may occur more often than supposed, considering that I found two such cases within one year—and without the use of the nasopharyngoscope. It is probable that some nasopharyngeal tumors originate in one tube.

2. Such tumors should be suspected in cases of unilateral catarrhal otitis, particularly of the secretory type, which do not respond to treatment.

3. Early diagnosis rests on repeated inspection of the tubal orifices, by both posterior rhinoscopy and nasopharyngoscopy.

4. Both cases reported were Grade IV squamous cell epitheliomata.

5. The one case personally treated, responded well to radium emanation and was apparently cured.

905 ROOSEVELT BUILDING.

The Scientific Papers of the American Broncho-Esophagological Association

LXXXVIII

PRESENTATION OF BIOPSY FORCEPS

SAM E. ROBERTS, M.D.

KANSAS CITY

I had these biopsy forceps made because I had difficulties with the ordinary straight biopsy forceps, especially the ones with the receding upper jaw. The tumor would be pushed downward and away from the forceps by the projecting lower jaw. With this forceps I am able to put it right on the tumor mass and get a good sized bite without any displacement of the tumor (Fig. 1).

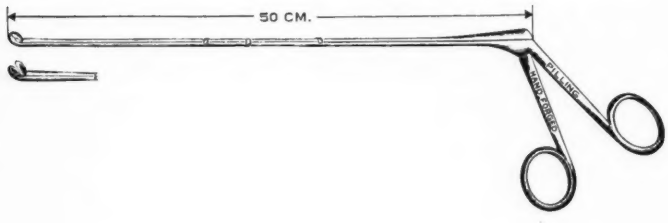


Fig. 1.

This, as you see, is an angle forceps very similar to the laryngeal biopsy forceps. This one worked so nicely I am now having a straight biopsy forceps made.

1103 GRAND AVE.

LXXXIX

PRESENTATION OF NEW INSTRUMENTS

SIMON JESBERG, M.D.

LOS ANGELES

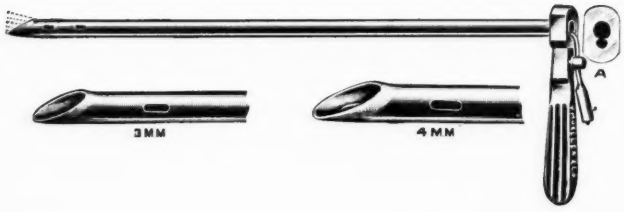
Two Baby Bronchoscopes: These instruments are designed with the object of having the greatest possible lumen and a minimum outside measurement. This is made possible by the use of the new miniature light bulb which is one millimeter less in diameter than the former standard size. In the smaller tube, which is three millimeters, the tunnel for the light carrier is open on top, thus giving more space. The lumen is sufficiently large to pass the Jackson forward grasping forceps. This tube can be used on the new-born. Its outside measurements are 5 x 4.5 millimeters, the length is twenty-six centimeters.

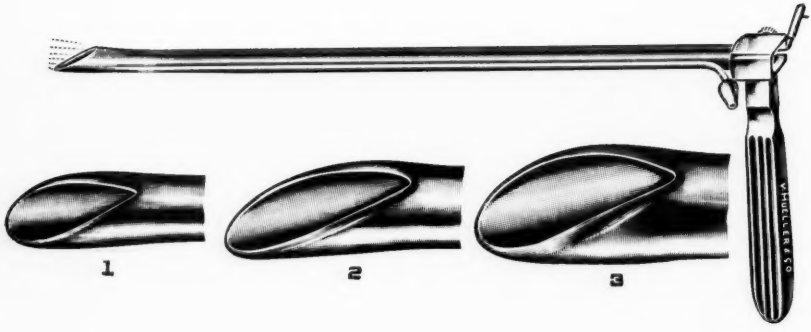
The four millimeter tube has a lumen the same size as the standard Jackson four-millimeter bronchoscope. Its outside measurements are 6.5 x 5.5 millimeters, as compared with the standard Jackson bronchoscope which has outside dimensions of 8 x 6.5 millimeters. It has been passed on the new-born cadaver but it is questionable if it could be safely used on the living child of that age. It was passed easily on a child nineteen days old. Its purpose is principally for the infant in which the subglottic lumen is narrowed by swelling, as is so frequently encountered in foreign body cases, when passage of a standard size tube is almost certain to necessitate tracheotomy (Fig. 1).

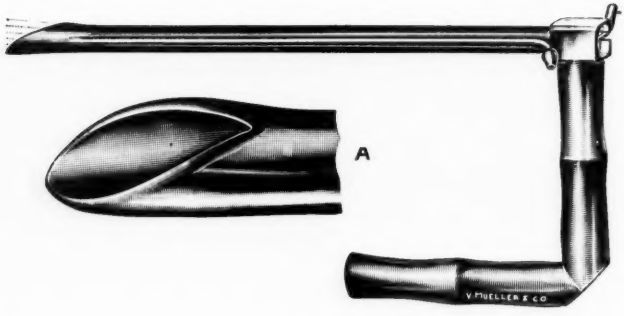
Esophagoscopes: For twelve years, I have been using esophagoscopes which are distally lighted and which have the distal tip modeled after the Haslinger instrument. Recently Mr. Mueller perfected the shape of these tips. I recommend them as being particularly safe in passage and easy to use. The special design protects the esophagus from trauma.

There are two adult esophagoscopes, one fifty centimers long, and the other, an upper esophagoscope, twenty-nine centimeters in length. Both have outside measurements of 17 x 12 mm. The

*Read before the twenty-third annual meeting of the American Broncho-Esophagological Association, New York, N. Y., June 5, 1940.


A
3MM
4MM


1
2
3
V.MUELLER & CO.


A
V.MUELLER & CO.

adolescent tube is forty-one centimeters long with outside measurements 15 x 9 mm. The child's esophagoscope measures thirty-two centimeters in length and has outside dimensions of 12 x 7 mm. (Fig. 2).

Bronchoscope Face Mask: This is composed of a transparency which is supported before the face, attached to spectacle frames (Fig. 3). The anterior surface has cells which accommodate trial case lenses. These lenses can be easily interchanged over the operator's head by an assistant. The lenses may be plano or contain a presbyopic correction. They are taken out of hot water, which both cleans and warms them. This mask offers complete protection for the bronchoscopic operator and affords him good vision.

500 South Lucas Ave.

XC

ANESTHESIA FOR PERORAL ENDOSCOPY

CHEVALIER L. JACKSON, M.D.

AND

GEORGE S. MCREYNOLDS, JR., M.D.

PHILADELPHIA

There are three outstanding requirements for a technic of peroral endoscopy, which may be summed up in the words safety, efficiency, and comfort. If these endoscopic procedures are to be more and widely accepted and approved by the physicians, surgeons and family doctors, they must first of all not jeopardize the patient's life; secondly, they must accomplish quickly and conclusively the diagnostic or therapeutic purpose for which they are done; and thirdly, they must be carried out with a minimum of pain and discomfort. We believe that proper attention to anesthesia will contribute substantially to the fulfillment of all three of these requirements.

There are a number of references to this subject in the literature, but we should like to mention particularly the paper read by the late Edwin McGinnis[1] of Chicago before this society in 1929. Though this paper is not published in full in the Transactions, the discussion by the members of the Society is very interesting indeed. We should also like to remind you of the communication of Dr. David Jones[2] read at the 1937 meeting, which was likewise followed by some instructive discussion, especially bearing on toxicity and means of preventing and treating its effects. In this connection we should like to mention also a very wise and helpful article by Henry Hall Forbes[3] on the use of local anesthesia, the paper by Hall and Thomas,[4] and the chapter dealing with local anesthesia in the concise and practical textbook of Imperatori and Burman.[5] An excellent paper on the Diagnosis and Treatment of Cocaine and Novocaine Reactions has recently been presented by Whayne Webb,[6] with a very complete bibliography. This important phase of the subject will not, however, be considered in our paper. Another good paper discussing the problem of anesthesia for peroral endoscopy is that of Putney.[7]

In beginning the present study, we believed that it would be especially interesting and profitable to make a survey of the methods of anesthesia practiced by the members of the Society and the opinions of the members regarding the various drugs in current use for premedication and anesthetization, both local and general. We therefore prepared a questionnaire, and I am happy to say that the response was even better than we anticipated; without any follow-up letters, we received replies from over eighty per cent of the members.

To summarize the replies to the questionnaire, let us consider first the replies on the subject of premedication, then the outline of technics used for the different endoscopic procedures, the opinions regarding the various drugs used for local anesthesia and finally the opinions and practice regarding the use of general anesthesia.

Morphine, with or without atropine, has long been regarded as the standard premedication, and it apparently still holds first place, being preferred by twenty-one of sixty some answering this question. Eleven used it with atropine, four with scopolamine, and six alone. One used it intravenously. Pantopon was used by three, and codeine by three. Nembutal was the next drug in popularity after morphine, though it was used alone by only five. Thirteen used it followed by morphine, and two followed by codeine. Sodium amytal was used alone by three, and with morphine by three others; sodium pantobarbital by two, and seconal by three. Two used dilaudid. Two stated that they did not use premedication.

Now let us review the technic of anesthetization used by our correspondents for the various endoscopic procedures. Since the great majority stated that they did not use general anesthesia or used it only on very limited indications, we shall defer consideration of it until later. For direct laryngoscopy, thirty-eight, or about fifty per cent, used swab applications to the pyriform sinuses, and thirty-nine used some sort of spray or instillation, but many of these used both, and therefore there was considerable overlapping in these groups. Five used a brush for application. Two used nerve-block. For bronchoscopy, fifty-eight stated that they used the same technic as for direct laryngoscopy, and thirty-nine used the same technic with the addition of the intratracheal instillation. For esophagoscopy, twenty-four stated that they used the same technic as for direct laryngoscopy and bronchoscopy. Four stated that they had the patient gargle the anesthetic solution. One used avertin rectally. Five used no anesthetic for esophagoscopy. For gastroscopy most used the same technic as for esophagoscopy, but twenty-four stated that they did not do gastroscopy.

In connection with technics of anesthesia for bronchoscopy, we should like to mention the suggestion recently made by Nail,[8] that the anesthetic solution be instilled through an intratracheal catheter. This offers a certain method introducing the solution and eliminates the necessity of supplementary application through the bronchoscope.

The various opinions on the advantages and disadvantages of the different drugs used for local anesthesia and also the opinions on avertin, evipal and pentothal sodium given in the replies to our questionnaire have been tabulated and summarized. Cocaine was still the preferred drug of forty-five (over 50%) though eight stated that they did not use it at all. Pontocaine was the next in popularity, being the preferred drug for local anesthesia in seventeen instances. Larocaine was preferred by five. Other local anesthestic agents mentioned by one or two of our correspondents were butyn, metycaine, metrozal, euphagin and Forestier's solution.

Avertin was approved by sixteen, condemned by fourteen, and "not used" by seventeen. Evipal was approved by thirteen and condemned by eighteen, while twenty-eight had not used it. Pentothal sodium had not been used by forty-four, but it is interesting to note that of those who had used it, the number approving it just about equalled the number condemning it (eight approving and seven condemning).

To conclude our survey of the experience and opinion of others, let us consider the views on general anesthesia. Seventeen did not use it at all, nine for children only. It was interesting to note that a number of individuals seemed to feel that general anesthesia was more necessary for esophagoscopy than for the other procedures. Three used general anesthesia for all esophagoscopies, while nine used it only for esophagoscopy in cases of large sharp foreign bodies.

The remainder of the paper will be devoted to outlining our current practice in the Temple University Clinic.

Premedication as a rule consists of an appropriate dose of morphine and atropine, and in especially apprehensive patients or in patients known to have a tendency to sensitivity to the local anesthetic drugs, a preliminary dose of nembutal may also be given, but this has not been routine. Sometimes a dose of nembutal is given the night before, and this dose repeated in the morning. The usual dosage of morphine is: morphine sulphate gr. 1/4 for the average

adult, with gr. 1/150 of atropine sulphate; for children of two years and over, the dosage is computed according to Young's rule ($\frac{\text{age}}{\text{age} + 12}$ = fraction of adult dose). For asthenic, thick-necked adult male, gr. 3/8 or 1/2 may be given, while for an underweight delicate adult female, gr. 1/6 or 1/8 would be sufficient. For direct laryngoscopy and bronchoscopy the atropine is thought to be as important as the morphine, because the lessening of salivation is very advantageous. Patients having a series of endoscopic treatments generally are not given premedication after the first few times, especially if treated as out-patients. In connection with premedication it seems appropriate to say that perhaps more important than any drug is what Dr. Chevalier Jackson, Sr., has called the "sermon on relaxation."[9]

As for the technic of anesthetization preliminary to the various procedures, let us consider first preparation for direct laryngoscopy and bronchoscopy in adults. It is our practice to use a spray to the pharynx, particularly the right side and the right anterior pillar, and then to follow with a fractional instillation into the larynx and trachea, under the guidance of the laryngeal mirror. Various drugs are used, and they will be discussed subsequently. The essential features of this technic are accuracy of placement and fractionation. Either a syringe such as that used by Lukens or an atomizer with a curved tip may be used, but the solution must get into the larynx and it must be fractionated into at least two or three instillations, a few drops at a time. For supplementary anesthesia of the bronchus, applications of a concentrated solution or an intrabronchial spray through the bronchoscope are used. Formerly swab applications to the pyriform sinuses by means of small gauze sponges held in "cross-action" forceps were used, but we believe that the method above described is much less disagreeable for the patient and even more efficacious.

For esophagoscopy in adults we generally use a simple spray to the pharynx, again taking care to favor the right side and especially the right anterior pillar. The patient is asked to gargle with the excess and spit it out. Occasionally, in the case of patients who have a tendency to cough during esophagoscopy, a few drops are instilled or sprayed into the larynx and the pyriform sinuses.

For gastroscopy with the Schindler flexible gastroscope we believe that the best results are obtained by fractional instillation with the ingenious Schindler anesthetizing tube,[10] though, of course, satis-

factory work can be done with the same simple preparation that is used for esophagoscopy.

In children under eight years of age, we do not as a rule use any anesthetic, local or general, though we believe that at least some of the drugs used for local anesthesia are quite nontoxic for children. As a matter of fact, the spraying or instillation of local anesthetics seems very often to annoy and distress a child more than the endoscopic procedure itself. One related matter that might be mentioned here is the particular importance of teamwork in the quick efficient endoscopic work necessary in unanesthetized children. A sheet should be laid on the table and the sheet folded over each arm separately and tucked under the body. This makes it possible for one person to hold the child's body firmly so that a properly trained head-holder and a skilled endoscopist can get the necessary work done in a minimum time and with maximum efficiency. Endoscopic procedures in children should be made as brief as possible, and the child's attention should be gotten away from the procedure as soon as possible afterward. It has long been customary in our clinic to keep a supply of "lolly-pops" on hand so that every child can be at once rewarded for good behavior, even though his behavior may not have been perfect. This practice helps to build up the child's morale and smooth the way for subsequent work. It applies especially to cases where a prolonged series of treatments will be required, rather than to the acute case such as the removal of a foreign body.

Cocaine is admittedly the most universally efficacious of the drugs used for local anesthesia, and if used with care, serious reactions will be rare. We still use it quite often, generally in 10 per cent solution, but we have also used larocaine 10 per cent. Pontocaine gives a fairly satisfactory anesthesia in the majority of cases, but it has several objectionable features. It is contraindicated in asthmatic patients because in these individuals it will frequently precipitate an attack, or at least produce slight wheezing. Another objectionable feature is the tendency to cause an irritation which may persist for several days. In spite of these objectionable features, we use this drug extensively, except in asthmatic patients. Larocaine has, in our experience, been absolutely nontoxic, but it has also been less efficacious, requiring a longer time to obtain maximum effect, and not giving as complete an anesthesia. We use it particularly in children.

General anesthesia is used in our clinics only very rarely, generally at the insistence of a patient or referring physician, but

occasionally by our own choice, as in the case of a small vocal nodule in a child or an apprehensive adult. In these latter cases, the use of general anesthesia is justifiable because of the importance of absolute fixation of the cord and the greatest accuracy of removal. Chloroform and ether have both been used occasionally in such cases. Avertin generally does not give sufficient relaxation or abolition of reflexes, certainly not unless given in full dosage and supplemented by local anesthesia. We use it routinely for esophagoscopically-guided diverticulectomy, but even in these cases the relaxation which would be ideal for the esophagoscopic part of the procedure is rarely obtained.

In connection with general anesthesia, it should be emphasized that while in many kinds of cases requiring endoscopy there would be no objection to its use, there are many other cases in which general anesthesia increases the risk enormously, and it therefore seems best to train one's self and one's team to do the work routinely without general anesthesia, if for no other reason than to avoid feeling at a disadvantage when confronted with a difficult case in which dyspnea, the presence of acute tracheo-bronchial inflammation, or some other condition contraindicates its use.

SUMMARY

The reasons for giving particular attention to the subject of anesthesia for peroral endoscopy are stated. The result of a survey of current practice and opinion, particularly among the members of the American Broncho-Esophagological Association, are then summarized. Finally the authors' own opinions are given, and an outline of their current practice presented.

255 S. 17TH STREET.

REFERENCES

1. McGinnis, Edwin: The Question of Anesthesia in Peroral Endoscopy. Tr. Amer. Bronchoscopic Soc., 1929.

2. Jones, David: Cocain Sensitivity in Bronchoscopy. Tr. Amer. Bronchoscopic Soc., 1937; also ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 46:749 (Sept.), 1937.

3. Forbes, Henry Hall: A Guide in the Use of Local Anesthesia. Eye, Ear, Nose and Throat Monthly (Feb.), 1926.

4. Hall, Sobisca S., and Thomas, H. V.: Local Anesthetic Crises in Rhino-Laryngology. W. Va. M. J., 34:516 (Nov.), 1938.

5. Imperatori, Charles J., and Burman, Herman J.: Diseases of the Nose and Throat, textbook, second ed., p. 576.

6. Webb, Whayne: Diagnosis and Treatment of Cocaine and Novocaine Reactions. (Paper read at meeting of Philadelphia Laryngological Society, May 7, 1940.) To be published.

7. Putney, F. Johnson: Anesthesia in Peroral Endoscopy. Laryngoscope, 49:55 (Jan.), 1939.

8. Nail, J. B.: Anesthesia in Peroral Endoscopy. Texas State J. of Med., Vol. 36 (Oct.), 1940.

9. Jackson, Chevalier, and Jackson, Chevalier L.: Bronchoscopy, Esophagoscopy and Gastroscopy, 3rd ed., W. B. Saunders Co., 1934.

10. Schindler, Rudolf: Gastroscopy with the Flexible Gastroscope. Textbook, University of Chicago Press, 1937.

XCI

DYSPNEA DUE TO CONGENITAL ANOMALY OF THE AORTA*

LeRoy A. Schall, M.D.

AND

Louis G. Johnson, M.D.

Boston

A white female infant, six months of age, was first seen on December 23, 1939, because of difficulty in breathing of ten days' duration.

The baby was full term following a normal delivery. She was nursed for one month and then placed on an adequate formula. She was apparently perfectly well until two weeks before admission, when she developed an upper respiratory infection. On the third day of this illness, she began to breathe noisily and her voice was almost gone. She developed a slight cough, which was treated as a bronchitis but which persisted until the time of admission. On two occasions she turned blue, stiffened with an arch back, and screamed for about one minute. She also made a peculiar rattling noise in her throat, especially at night, and wheezing was constant but varied in degree.

Careful questioning of the mother brought out the history that from the age of two months the baby assumed an unnatural position while sleeping, the head being thrown in extreme extension at almost right angles to the shoulders, and that she made a peculiar noise while breathing, particularly during the night.

Physical examination revealed a baby with a noisy, rattling breathing, who on trying to cry uttered only a short thin squeak. On crying hard the face and lips became cyanotic. There was a considerable amount of mucus in the pharynx. The chest moved normally, and the lungs were well aerated throughout.

---

*Read before the twenty-third annual meeting of the American Broncho-Esophagological Association, New York, N. Y., June 5, 1940.

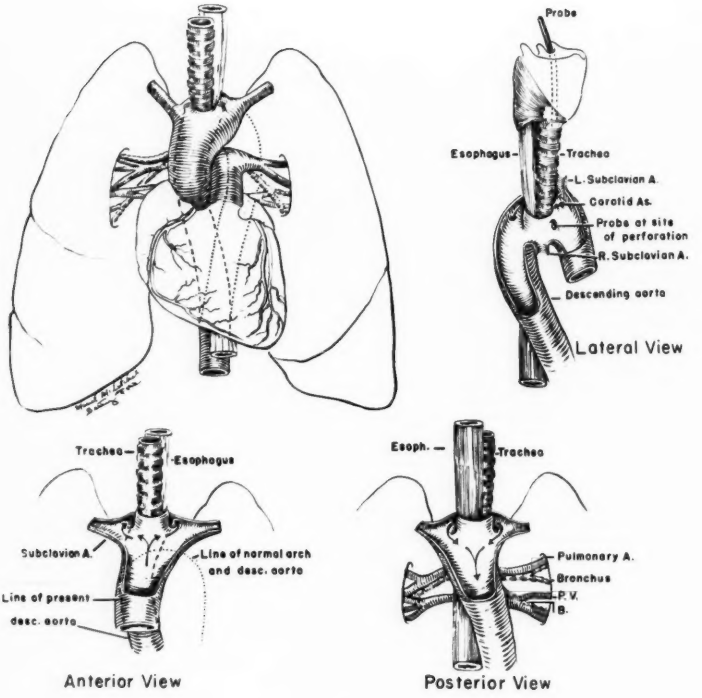


Fig. 1.

X-rays and fluoroscopy of the chest showed normal movements of the diaphragm. There was no evidence of metallic or vegetable foreign body. Direct laryngoscopy showed a slight redness on the posterior surface of the arytenoids without swelling.

The vocal cords were normal in color without paralysis. There was no evidence of laryngeal obstruction.

The patient was sent home to await developments but was readmitted in sixteen days with the persistence of the same complaints.

Again the larynx was examined with absolutely the same findings. Bronchoscopy was performed using a 4 mm. Jackson bronchoscope, and the only pathological finding was a slight constriction of the trachea just above the carina. This constriction had the

appearance of a slightly swollen mucous membrane. There was no pulsation. With the bronchoscope past the constriction, breathing was very quiet and easy. Following the removal of the bronchoscope the child became cyanotic with difficult respirations. The bronchoscope was reinserted and a low tracheotomy performed.

During the next two weeks the child ran a remittent fever ranging from 99 to 104 degrees, with a leukocytosis up to 21,000 with varying degrees of atelectasis of the left lung. Cultures from the trachea showed no growth. During this time she developed respiratory difficulty with cyanosis which necessitated bronchoscopy, and it was noted that as soon as the bronchoscope reached the carina the cyanosis was relieved and the breathing was easier. At each bronchoscopy very minute crusts were removed which, too, helped to improve the breathing. Bronchoscopies became increasingly more frequent. With the bronchoscope in place the baby could breathe. As soon as it was removed there was great difficulty. Finally a cane tracheotomy tube which reached to the carina gave relief. With this tracheotomy tube the baby was in no discomfort, perfectly happy and played about the crib.

Ten days later, without any warning, she suddenly began to cough up bright red blood followed in a matter of second by profuse hemorrhage through the tracheotomy tube and through the mouth, and rapidly died from hemorrhage.

POSTMORTEM EXAMINATION

Postmortem examination showed a well-developed, well-nourished six-months-old white female child, measuring 66 cm. in length and weighing about 20 pounds. In the midline of the neck there was a tracheotomy wound measuring 1 cm in diameter, the opening of which was not obstructed. Upon moving the body a small amount of dull red blood issued from the mouth.

The larynx appeared to be slightly edematous, but the vocal cords were in the cadaveric position and apparently permitted free passage of air. There was a tracheotomy wound measuring 1 cm. in diameter on the anterior surface of the trachea immediately below the cricoid cartilage. The trachea appeared to be slightly constricted at a point about 1 cm. above the carina. The mucosa was bright red, soft and edematous.

The lungs were pale whitish-pink and apparently well aerated. Throughout the right lung there were a few dark red focal areas of

apparent intrapulmonary hemorrhage, measuring up to 1 cm. in diameter.

The heart showed hypertrophy of the ventricular walls, especially the left, the measurements being: right, 5 mm., and left, 10 mm. in thickness. The right ventricle was comparatively small and did not extend to the apex of the heart, its left border being 1 cm. to the right of this point. The left ventricle was correspondingly large and consequently formed the major portion of the heart. The foramen ovale was patent.

There was a persistence of both embryonic fourth arches, forming a left and a right aortic arch. These two arches took their origin from the common trunk of the ascending aorta 2.5 cm above the aortic valve at a point to form the common descending aorta immediately posterior to the esophagus. The two arches together formed a circle enclosing within it the trachea and esophagus and producing the previously described slight narrowing of the trachea 1 cm. above the carina. There was, however, no apparent constriction of the esophagus, and gas could be freely passed from one end to the other. Both aortic arches were patent throughout their entire length and each measured 1.3 cm. in circumference, while the circumference of the descending aorta was 1.6 cm. The left carotid and subclavian arteries arose from the left aortic arch in the usual manner. The innominate artery, however, was absent and in its stead the right carotid and subclavian arteries arose as separate entities from the right aortic arch, thus presenting a symmetrical right and left sided circulation. On the medial surface of the right aortic arch as it lies in relation to the trachea, there was a minute irregularly linear erosion measuring 0.3 cm. in length, which communicated with the lumen of the trachea.

The pulmonary artery lay in its usual relationship to the surrounding structures, and the ductus arteriosis was absent.

DISCUSSION

This case of double aortic arch is illustrative of one of the rarer forms of congenital heart disease. Dr. Maude Abbott[1] reports five such cases in an analysis of one thousand congenital cardiac anomalies, and Hermann[2] states that twenty-one cases have been reported up to 1928.

A brief review of the comparative anatomy and embryology of the aortic arches throws some light on the formation of this

developmental anomaly. Of the six or more aortic arches present in the fishes, only the fourth pair persist in complete form in certain reptiles. These unite to form the dorsal aorta. In birds only the right fourth arch is retained, while in mammals the left forms the normal aortic arch. Portions of the other arches, however, do persist normally in mammals. Thus only the distal portion of the right fourth arch is obliterated, while the proximal portion forms the innominate artery. In the case under discussion this obliteration of the distal portion of the right fourth arch has not occurred, but instead, as in reptiles, the whole arch has persisted, and together with the left arch completely surrounds the trachea and esophagus in a vascular ring. If the above hypothesis is true, one would expect to find all degrees of persistence and absence of the aortic arches. This is found to be the case. In some instances, for example, the left arch is entirely absent or else remains as an obliterated fibrous cord. In other cases one trunk may be smaller than the other, etc., a variety of such combinations having been reported.

The associated hypertrophy of the left ventricle is a matter for conjecture. In Dr. Abbott's[1] series, one case had hypertrophy of the left ventricle and one hypertrophy of the right ventricle. In this case the hypertrophy might possibly be due to a relative coarctation of the aorta since each arch measured 1.3 cm. in circumference while the circumference of the descending aorta was only 1.6 cm.

Of the other cases of double aortic arch reported in the literature, some have occurred without symptoms, one patient living to the age of 86 years. Others are associated with various pressure symptoms, dysphagia being prominent. Symptoms and signs due to pressure on the trachea, such as occurred in this case, appear to be less common. A case reported by Hermann,[2] however, had strikingly similar symptomatology. This was a six-months-old child with peculiar wheezing respirations, cyanosis on exertion, and a short staccato cough like the bark of a dog.

The diagnosis, especially if unsuspected, is difficult during life. However, the similarity of the symptoms, namely, peculiar wheezing, cyanosis on exertion, and staccato cough, in the two above mentioned six-months-old children should be emphasized. In such cases when no other cause can be found, the possibility of some anomaly of the aortic arches should be considered. Sprague, Ernlund and Albright[3] have shown the importance of and enumerated the points in the radiologic diagnosis of persistent right aortic arch. Visualization of the esophagus with a barium meal in cases of double

aortic arch should show uniform constriction of the esophagus on all sides, whereas in the normal aortic arch it is deflected to the right.

243 CHARLES STREET.

BIBLIOGRAPHY

1. Abbott, M. E.: Congenital Cardiac Disease. Atlas of Congenital Cardiac Disease.

2. Hermann, W. W.: Double Aortic Arch. Arch. of Path., 6:419, 1928.

3. Sprague, H. B., Ernlung, C. H., and Albright, F.: Clinical Aspects of Persistent Right Aortic Root. New England J. of Med., 209:14:679.

XCII

CENTER OF OSSIFICATION OF THE STERNUM MISTAKEN FOR A FOREIGN BODY IN THE ESOPHAGUS*

ERNEST M. SEYDELL, M.D.

WICHITA

Before giving my case report I think it may be well to refresh your memory in reference to the ossification of the sternum. According to Morris' Anatomy the ossification of the sternum is slow and irregular. "The process begins in the manubrium by a single center about the sixth month of intra-uterine life, though occasionally other accessory centers are superadded. The body usually ossifies from seven centers. The upper segment ossifies from a single median nucleus about the eighth month, and below this three pairs of ossific nuclei appear which may remain for a long time separate. Of these, two pairs for the second and third segments are visible at birth, and those for the lower segment make their appearance towards the end of the first year. The various lateral centers unite in pairs, so that at the sixth year the sternum consists of six pieces, the lowest (xiphoid) being cartilaginous. Very often, however, there are only four centers of ossification in the body. Gradually the four pieces representing the body fuse with one another, and at twenty-five they form a single piece, but exhibit even in advanced life traces of their original separation."

REPORT OF A CASE

A child, M. W. C., 14 months of age, was brought to me for consultation by his physician, Dr. Paul Carson, on July 11, 1939, on account of a severe cough and dyspnea, and the following history was obtained.

The child had had recurring attacks of croupy cough since birth. In December 1938 he developed a pneumonia followed by a prolonged convalescence which was characterized by a continuous cough and fever. Preceding this illness he had choked rather severely while playing with a celluloid toy, and the parents suspected that he might have swallowed or inhaled a portion of it.

*Presented before the annual meeting of the American Broncho-Esophagological Association, New York City, June 5, 1940.

The present illness began about two and one-half weeks prior to my examination with a slight head cold followed by a croupy cough and fever. He had refused all foods with the exception of liquids during the last two weeks. His cough had grown gradually worse until he now coughed almost continuously. Dyspnea had developed during the last twelve hours. The child had been fluoroscoped by Dr. Carson, but no foreign body could be seen. In view of the fact that the child had a constant croupy cough and dyspnea and had refused solid food, it was thought advisable to make a direct examination of his respiratory tract. A laryngoscopic examination was therefore carried out. The laryngeal findings were negative with the exception of a slight redness of the vocal cords. A three and one-half millimeter bronchoscope was now introduced into the trachea and considerable resistance was met with in the subglottic area. There were no other pathologic findings. The child was returned to his room, and later a roentgen ray study was made of his chest. Much to our surprise we received the following report from our radiologist, Dr. Frost. "In the upper thoracic region just below the sternoclavicular joint on the left side is an annular shadow about the size of a quarter. It persists on several examinations and is of the density of a paper, thin metal, or a composition of some type. The definite location is not determined."

On the following day I decided to examine his esophagus, and again I could locate no foreign body. His breathing became more and more difficult and cyanosis increased. On July 14, 1939, I was obliged to perform a tracheotomy. There was considerable postoperative fever for several days, his coughing was relieved, and his appetite returned. On July 23, 1939, I replaced the Number Two tracheotomy tube with a Number One; following this I inserted partial plugs in the inner tube. As soon as the child was forced to breathe though his larynx his cough returned. We were unable to remove his tracheotomy tube until July 31, seventeen days after its insertion.

Following our esophageal examination, Dr. Frost checked several hundred x-ray films of children and found disc-shaped shadows in eight other cases. It then became obvious that the shadow we had mistaken for a foreign body was in reality a germinal center of the sternum.

In conclusion I wish to state that in this case we undoubtedly had a congenitally small subglottic space which became edematous following a respiratory infection or allergic upset, the latter statement being based on the fact that this winter he again developed a

croupy cough which was promptly relieved by the administration of Tedral.

Soon after this little patient returned to his home, his mother gave birth to twins. One twin when several months of age developed a head cold and subsequently a croupy cough identical with that of his older brother.

107 W. Douglas Ave.

XCIII

BRONCHOSCOPY IN TUBERCULOUS CHILDREN

MERVIN C. MYERSON, M.D.

NEW YORK

Bronchoscopy is an accepted diagnostic and therapeutic procedure in the management of the tuberculous patient. Much has been written about tuberculous lesions which have been observed in adults. Except for the report of an occasional unusual and unexpected bronchoscopic experience, nothing has been written on the subject of bronchoscopy among tuberculous children.

During the past two years 29 tuberculous children have been bronchoscoped. The ages ranged between 5 and 16 years. They were referred for the following reasons:

1. Unexplained atelectasis.

2. To obtain bronchial smears when the feces and gastric secretion were negative for tubercle bacilli.

3. To localize the active lesion in the lung.

4. Because of a positive sputum in the presence of an apparently successful pneumothorax.

5. Noisy breathing.

There were thirteen positive bronchoscopic findings among the twenty-nine patients. They were distributed as follows:

Indentation by enlarged glands	7
Perforation by tuberculous gland	1
Absorption of cartilages and collapse of wall	1
Atelectasis due to secretion	3
Atelectasis due to ulcerogranuloma	1
	13

The presence of the tubercle bacillus in the bronchial secretion was established in nine cases. A single bronchoscopy was sufficient

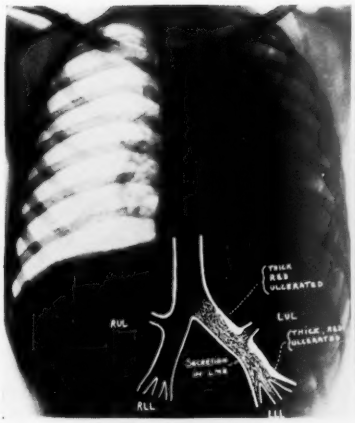


Fig. 1.

Fig. 2.

Fig. 1, Case 1. Roentgenogram showing massive atelectasis of left lung due to retained secretions.

Fig. 2, Case 1. Roentgenogram of chest immediately after bronchoscopic removal of obstructing secretions.

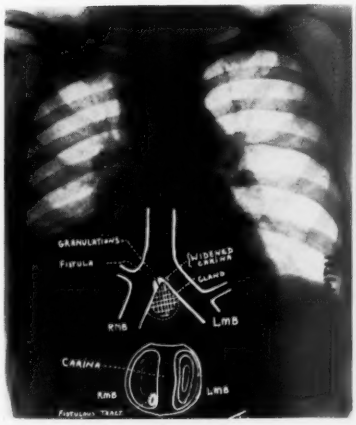


Fig. 3.

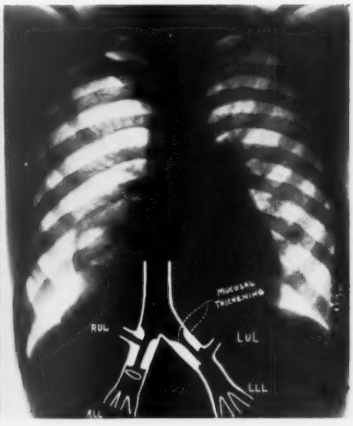


Fig. 4.

Fig. 3, Case 3. Roentgenogram and diagram showing condition of chest and fistulous tract from mediastinal gland into right main bronchus.

Fig. 4, Case 5. Roentgenogram and diagram showing narrowing of main bronchi due to submucous infiltration.

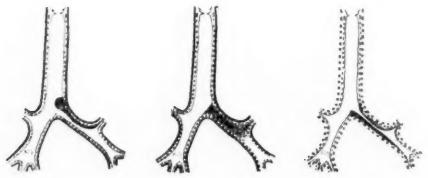


Fig. 5, Case 2. Representation of conditions in the bronchial tree showing progress of lesion until terminal stage of fibrosis with a reformed lumen. First drawing indicates condition in October 1938; second, February 1939; third, July 1939.

to establish the presence of organisms after numerous sputum and gastric examinations had proven negative.

The following cases are of interest:

CASE 1.—An 11-year-old colored female was admitted in May, 1939. Dullness and numerous coarse rales were found over the left chest. The sputum and gastric content contained tubercle bacilli. In May the x-ray film revealed "an irregular massive density involving the left lung which appeared to be airless. The heart and mediastinum are retracted markedly to the left." Bronchoscopy was requested to establish the cause of the massive atelectasis.

At bronchoscopy a large amount of viscid mucopurulent secretion was found filling the bronchus. This was removed by aspiration. The introitus of the left main bronchus was red and thickened. Immediately after evacuation of the thick secretion an x-ray study of the chest was made. This revealed a considerable degree of reaeration of the lung, with evidence of the presence of several cavities. The atelectasis had been caused by the secretion. (See Figs. 1 and 2.)

CASE 2.—A 14-year-old white girl was admitted to the hospital in August, 1938. Artificial pneumothorax had been used to induce collapse of the left lung before admission. Despite what appeared to be a successful collapse, the sputum remained positive for tubercle bacilli. A month after admission bronchoscopy disclosed a large ulcerogranuloma on the anterior and lateral tracheal walls just above the carina. A similar lesion could be seen on the spur dividing the left main bronchus from the upper lobe branch. Some mucoid material was seen entering the main bronchus from the upper lobe branch. A smear made from this material revealed the presence of tubercle bacilli.

During a second bronchoscopy in October, the granulomatous mass previously noted was no longer present; instead an ulcerogranulomatous process extended from the carina into the main bronchus and disappeared into the lumen of the upper lobe bronchus. In January the patient developed evidence of obstruction of the left main bronchus, with a high degree of negative pressure in the left chest, and a shift of the mediastinum toward the involved side. After this, all examinations for tubercle bacilli were negative.

A third bronchoscopy was performed in February. The lumen of the lower half of the [illegible] was narrowed to about ten millimeters by an ulcerogranuloma [illegible] extended almost to the carina. From the lower limit of this region there extended an early fibrotic lesion along the left tracheal wall and across the introitus of the main bronchus. As a result, neither the left bronchial lumen nor the left side of the carina could be seen. Only a single passage existed, that between the trachea and the right main bronchus.

Just before her discharge from the hospital, in July, an x-ray film revealed that the left lung had re-expanded; this meant that a lumen had been re-established in the left main bronchus. Unfortunately, bronchoscopy was not performed at this time. (See Fig. 5.)

This was the first child in whom we were privileged to observe all the changes of a tuberculous bronchial lesion.

CASE 3.—A 12-year-old colored girl was admitted December 28, 1938, complaining of a cough and a daily evening rise of temperature. Examination revealed the presence of dullness and numerous rales in the lower half of the right chest. There was an area of increased density occupying the same region on the x-ray film. The hilar markings of the right side were exaggerated. The sputum and gastric content did not contain tubercle bacilli. Later x-ray study disclosed a slight retraction of the tissues of the right lower lobe and a small degree of deviation of the mediastinum to the same side. There was evidence of enlarged mediastinal lymph nodes. A compensatory emphysema of the left lung was present.

Bronchoscopy was performed early in February. The left bronchial tree was uninvolved; the carina was greatly thickened and widened. A fistula, surrounded by thick red granulation tissue, was found on the right side of the carina, extending obliquely downward and to the left. A disintegrating interbronchial gland had perforated into the point of juncture of the right bronchus and trachea. (See Fig. 3.)

CASE 4.—A 14-year-old white girl was admitted in June, 1939. She complained of chills, fever, sore throat and cough. Her illness had begun three months before. X-ray study revealed a circumscribed oval area of increased density in the left hilar region; this appeared to be due to a mass of glands. A similar area of density was seen in the right paratracheal region.

Bronchoscopy, which had been performed in another hospital in May, revealed a shortened and flattened carina. The left main bronchial lumen had been reduced to half its size by pressure upon its medial wall in the region of the carina.

Bronchoscopy performed shortly after admission demonstrated a bulge of the medial wall of the left main bronchus, just above the region of the upper lobe branch. The changes previously noted about the carina were no longer present.

CASE 5.—An 11-year-old colored girl was admitted to the hospital in February, 1939. Her illness began two months before, when she had a "cold" which persisted. At the time of admission she complained of productive cough, loss of weight and night sweats.

A hydropneumothorax of the right chest was demonstrated on the x-ray film in June. Anteriorly, in the region of the fourth interspace, a large cavity could be seen. There was a short period of quiescence of the child's disease, after which there was renewed activity and a return of tubercle bacilli in the sputum.

Bronchoscopy was performed early in July. Both main bronchi were concentrically narrowed from a point just below the carina, so that a 6-millimeter lumen bronchoscope could not be passed through them. This narrowing was considered the result of submucous infiltration with tuberculous elements. (See Fig. 4.)

CASE 6.—A 15-year-old colored boy began his illness in May, 1939, with cough, expectoration, loss of weight and chest pain. He was admitted to the hospital two months later; shortly after, a selective pneumothorax was performed upon his left chest. The x-ray study revealed a shift of the mediastinum to the right; an irregular triangular shadow was seen behind the left side of the cardiac shadow. Bronchial occlusion was suspected. A previous x-ray film, developed elsewhere, suggested the presence of enlarged mediastinal lymph glands, especially about the left main bronchus.

Bronchoscopy was performed four days after admission. The left main bronchus contained thick, odorless, greenish exudate. The

lumen presented as a semilunar slit because of an inward bulge of the posterior wall. The greatly reduced lumen, together with the presence of thick exudate in the bronchus, accounted for the temporary occlusion.

CASE 7.—A 14-year-old white girl became ill in March 1939. She had fever, chills, sore throat and cough. She was admitted to the hospital two months later, with evidence of disease in the right mid-lung field. There were no tubercle bacilli in her sputum. X-ray study demonstrated a discrete oval area of increased density in the left hilum which was thought to be due to a collection of enlarged lymph nodes.

Bronchoscopy revealed a shortened and flattened carina. The left main bronchial lumen was reduced to one-half its size by pressure upward of its medial wall in the region of the carina. This pressure was due to enlarged mediastinal glands.

CONCLUSIONS

The results of experiences in a series of 29 bronchoscopies among tuberculous children are given. The ulcerogranulomatous type of lesion frequently seen in adults is not as common among children. Instead, the most common gross lesion is that caused by pressure upon the bronchial wall of enlarged mediastinal glands. Perforation into the lumen of a tuberculous process originating in an adjacent gland is extremely uncommon, as is also absorption of cartilages due to external pressure.

Bronchoscopy furnishes the clinician with a better understanding of the chest of the tuberculous child.

136 E. 64TH ST.

XCIV

LARYNGOTRACHEOBRONCHITIS*

GEORGE RENFREW BRIGHTON, M.D.

NEW YORK

The treatment of this disease has been made very difficult because the diagnosis depends to a large extent on the condition of the larynx and tracheal mucosa after the disease has become established. If one were to diagnose all cases of laryngeal dyspnea as laryngotracheobronchitis the number of cases in the literature would increase tremendously. For my purpose in diagnosing this condition I have limited the diagnosis of laryngotracheobronchitis only to those cases in which the obstructive laryngeal dyspnea was sufficient to warrant tracheotomy.

In reviewing the literature an attempt has been made for purposes of comparison to take from the statistics of others only the cases which they report to have been tracheotomized, and the figures in the table (Table I) show that our mortality statistics are still very much the same as the early groups.

TABLE I

Name	Tracheotomies	Deaths	Tracheotomic Mortality	Date
Gittens	15	8	53%	1936
Smith	12	4	33%	1936
Baum	13	8	61%	1928
Richards	23	12	50%	1937
Brennemann and Holinger	24	10	42%	1936
Brighton	27	11	40%	1940

The accompanying graph shows the incidence of cases by years in this series of laryngotracheobronchitis (Fig. 1). The criterion for the diagnosis was obstructive laryngeal dyspnea which by laryngoscopic examination revealed subglottic edema and which was severe enough to warrant a tracheotomy. In this group there were 27 cases

*Presented before the annual meeting of the American Broncho-Esophagological Association, New York City, June 5, 1940.

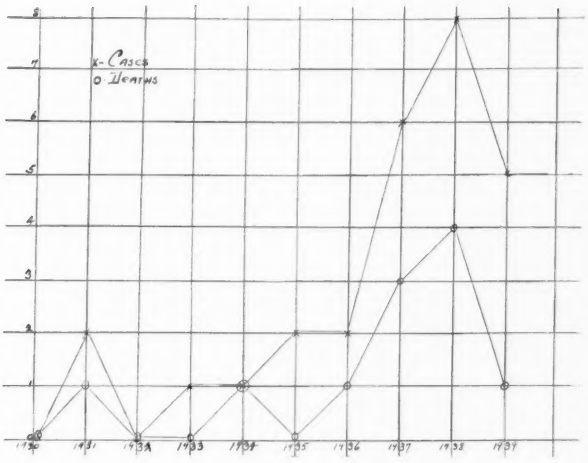


Fig. 1.

in all; 11 patients died, resulting in a mortality of approximately 40 per cent.

Table II shows the diversity of bacteria. In most of the cases there were several organisms found on the smear and in the culture. It seemed that there might be a symbiosis between the hemolytic streptococci and the hemolytic staphylococci aureus because the cases in which these two organisms were found together were usually the more severe. It can be seen also that the pneumococci occurred with great frequency at certain periods. However I do not believe that the bacteriology has given us much help or information in preparing for the treatment.

The average age of these patients was 27 months; the youngest child was 11 months old. There were 10 females and 17 males. The shortest interval from the onset of the disease to tracheotomy was four hours and the longest interval was 14 days. The average length of time that the tracheotomy tubes were left in place was 22 days. We were able to extubate one patient in 10 days. The longest period of time that a tube was worn in this series was 41 days.

It has been suggested by some of the recent authors that perhaps the advent of sulphanilamide or chemotherapy might change the course of our severe mortality. In this series 10 cases were given

adequate doses of sulphanilamide, and of these 10 cases six children died. While the series is not large enough to be convincing it appears that the chemotherapy has not influenced the mortality rate.

TABLE II

Case No.	Hemo. Strep.	Strep. Vir	Pneumo.	H. Infl.	Staph. Albus Hemo.	Staph. Aur. Hemo.
1.	X		X		X	
3.			X Type 7		X	
4.					X	
5.	X					X
6.	X					
7.						X
8.	X					
9.	X		X			
10.	X					X
11.						X
12.	X			X		
13.					X	
14.	X					
15.		X	X Type 19			
16.			X Type 19	X		X
17.			X Type 20			X
18.			X Type 19			
19.			X Type 18-19-23-4			
20.		X	X			
21.	X	X				
22.			X	X		X
23.		X				
24.		X		X		
25.			X Type 21			
26.				X Type B		
27.		X				

Before analyzing the 11 cases which died I think it might be wise to outline a routine treatment for obstructive laryngeal dyspnea. Immediately upon admission the child is placed in a room filled with

moisture and the respiratory movements observed. Frequently we use an oxygen tent, and a year ago we used oxygen and helium. We do not feel that the oxygen or oxygen and helium are substitutes for tracheotomy in these acute cases of obstructive laryngeal dyspnea, but we are able at times to forego an operation in cases of simple edema of the larynx and other conditions not associated with acute laryngotracheobronchitis. The patients are all given adequate sedation under the direction of the pediatrist in charge of the case. If it appears that respiratory distress is increasing a laryngoscopy is performed. Frequently, at the same time the child is fluoroscoped to determine the position of the mediastinum and whether or not there is a foreign body present. Following this a tracheotomy is performed. This has been the routine in all of the 27 cases in this group. During the last three years all of the cases have been given chemotherapy as a routine. After the tracheotomy is performed the child is returned to the room in which there is a high humidity and the respiratory movements very carefully watched, the child especially attended by a trained tracheotomy nurse and kept under surveillance by the pediatric resident and the resident in otolaryngology, who are also trained in the handling of tracheotomized patients. A suction machine is in constant attendance, and we have used a small rubber catheter with fairly firm walls attached to the suction machine.

In the past 10 years we have changed from a normal saline solution instilled into the tube before suction to a 1 per cent solution of ephedrine in saline before suctioning, and recently we have been using a 5 per cent sterile bicarbonate of soda solution instilled through the tracheotomy tube before the rubber catheter is placed into the tube and trachea just before the removal of secretions. We have found it advantageous when the physical signs show obstruction in one of the lobes of the lung to roll the child toward the involved side when the bicarbonate of soda solution is dropped through the tube. We have been able to remove much of the obstructive material in this way, and it seems to be less traumatic than bronchoscopic removal. We have avoided all of the opiates for sedation and used moderate doses of luminal. The supportive treatment of the child is kept under the careful surveillance of the pediatrist, and we have made it a definite rule that the child is not to be disturbed by frequent physical examinations or by procedures which may cause it annoyance. Since this disease is a very complicated problem it requires the cooperation of the nursing staff and the pediatric staff with the bronchoscopist. When a tracheotomy should be done is always discussed by the two groups because we feel that the condi-

tion of the heart and mediastinum should be carefully observed in an effort to avoid the accidents which sometimes occur when tracheotomy is too long delayed. In this series roentgenographic observations were made following tracheotomy to determine the position of the tube, and it was discovered that there were six cases out of the total in which there was a demonstrable emphysema of the mediastinum or a pneumothorax. It appears, therefore, that emphysema following tracheotomy occurs more frequently than many authors have led us to believe, but I do not think it influences the course of the laryngotracheobronchitis.

Inasmuch as we can gain more information from the study of our failures than our successes it seems advisable to analyze the 11 deaths in an effort to determine if there is any group of factors which might increase the number of successes. In the group of 11 deaths, five of these children died within 12 hours of admission, two of them died within three days of admission and six of them died not later than the seventh day after admission. It is interesting to note that not one of the fatal cases was left with its obstructive laryngeal dyspnea for more than 72 hours after the onset of the disease. The average time for the 11 cases was 27 hours. As we have said before, six of these cases had adequate doses of sulphanilamide, and six of the cases were bronchoscoped one or more times. In comparing this group with the children who survived the disease there does not seem to be any group of factors in the treatment which influences the course of the severe disease. It is perfectly possible that the only children in this group who had true laryngotracheobronchitis were the 11 patients who died, but I can say that the 16 cases which survived all had considerable suction with the catheter method described above, and many of them had a moderate amount of crusting. It is our opinion that the 16 cases which survived did so because of the immunity to a specific organism of which we will speak later.

In an attempt to formulate an idea as to a specific disease a comparison will be made between the pathology of a disease known as laryngotracheitis in chickens, first described by May and Tittsler in the Journal of the American Veterinary Association in 1925. This disease apparently has the same clinical manifestations in the fowl as the acute fulminating laryngotracheobronchitis in children. In the description of the pathology in infectious laryngotracheitis in chickens, J. R. Beach was able to show that the causative agent in the disease was contained in the exudate which accumulates in the larynx and trachea of infected chickens. By a series of bacteriological studies of the tracheal exudates he was able to determine that in spite

of the fact that there were innumerable organisms found in this exudate, by passing it through Berfeld filters and then inoculating other chickens with the filtrate he was able to cause the same type of disease. These studies of the exudate and its filterability showed that it contained a filterable virus. An attempt was made to inoculate sparrows, doves, crows, starlings, pigeons and ducks but these were unsuccessful although with the same filtrate the inoculated chickens died very rapidly. Beach felt that his experiments definitely proved that laryngotracheitis in chickens is due to a filterable virus. He was able also to prove that sera from fowls which had recovered from an infection with one strain will neutralize the same strain of virus.

Continuing the study of this disease from a standpoint of histopathology Oskar Seifried, from the Department of Animal Pathology of the Rockefeller Institute, studied 36 cases of laryngotracheoitis. Some of the fowls were killed with chloroform between six hours and seven days after inoculation while some others died in various stages of the disease. He described the gross pathology of the larynx and trachea as follows:

"*Larynx and Trachea.*—At autopsy the most striking lesions were regularly found in the upper larynx, the trachea, and less frequently the lower larynx. Six to 42 hours after inoculation only a small amount of mucus was visible. Between 42 and 74 hours after inoculation there might be small hemorrhages in the mucous membrane or a small amount of mucus mixed with clotted blood and restricted entirely to the upper larynx, while three or more days after inoculation the mucus might be replaced by caseous, yellowish masses which collected in the larynx or trachea to such an extent that they entirely plugged the opening. The mucous membrane of the lower trachea was normal except for a slight swelling, reddening, and increase of mucus which covered it with a thin film. In most of our cases this process extended the entire length of the trachea and often into the main bronchi. The degree and the kind of lesions varied greatly. Frequently the mucous membrane was covered with a comparatively thin film of clotted blood, with mucofibrinous and caseous masses or with a mixture of blood and exudate, while in other cases clotted blood or exudate was collected in a mass so large that it completely occluded the lumen of the lower portion of the trachea and of the main bronchi. Sometimes the amount of clotted blood exceeded that of mucus. Cross-sections through the larynx, trachea, and bronchi showed an enormous thickening of the mucous membrane.

Except for a more or less marked congestion and edema the lungs appeared entirely normal in most of our cases. In a very few a yellowish exudate between the chest wall in either or both lungs and foci of consolidation and necrosis of a yellowish color were present."

The histology of the trachea and larynx is described as follows:

"*Larynx and Trachea.*—24 to 72 hours after inoculation the predominant picture is a more or less marked edema and cellular infiltration of the tunica propria and the submucosa. In some of our cases the edema of the mucous membrane causes an entire disarrangement of its normal structure. The inflammatory process may even spread into the adventitia. One of the most noticeable lesions is the presence of small hemorrhages around smaller vessels of the propria mucosa and submucosa or such an extravasation of the red blood corpuscles that the mucosa and submucosa are completely separated from each other. A few red blood corpuscles also occur throughout the upper part of the edematous mucous membrane and even appear on its surface. In view of the negative histological findings in the walls of the blood vessels in these early stages, one is led to believe that the escape of cells is due to rupture of smaller vessels of the submucosa caused by violent coughing and respiratory efforts or possibly by diapedesis.

"In later stages of the disease (three to seven days after inoculation) the lesions in the larynx and trachea are characterized by a dense cellular infiltration in both tunica propria and submucosa. From the comparative study of a number of cases it becomes evident that the larynx as a rule shows more infiltration than the trachea and bronchi. While in some of the cases the normal structure of the mucous membrane and the mucous glands can still be recognized, in the majority there is such a dense infiltration that the epithelial elements become more or less dislocated and destroyed.

"The greater number of the infiltrating cells are small lymphocytes with histiocytes and few plasma cells. The proportion of the different kinds varies; while in some cases the lymphocytes are most numerous, the histiocytes predominate in others. Eosinophilic leucocytes, present normally in the trachea of chickens, may be increased in number. Mitotic figures indicate a slight increase of the connective tissue elements. In sections impregnated by Bielschowsky-Maresch's silver method connective tissue fibers are found to be increased. The fibers are swollen and sometimes broken as a result of the enormous edema. The same is true of the elastic tissue fibers.

The blood vessels show no distinct and regular lesions except for a more or less marked swelling of the endothelial cells and a change in the chromatin structure of the nuclei. In some cases retrogressive changes are found in the connective tissue cells of the submucosa which may contain small fat droplets in their protoplasm and nuclei. This degenerative process does not usually reach a high degree during the first five days after infection. Older cases however frequently show actual necrotic foci in the submucosa and tunica propria with a general hyperchromasia, pycnosis, and karyorrhexis of nuclei, and fatty degeneration of cells including those of the various infiltrating elements. In these late cases and sometimes also in earlier ones, a marked proliferation of endothelial cells with necrosis of the walls of the blood vessels is present. These lesions, when they occur, may provide an entrance for the virus into the blood stream and its conveyance to the liver and spleen in which, according to Beach, it is found frequently.

"While in some very early cases the epithelium of the mucous membrane and of the mucous glands retains its normal structure and the individual cells are almost normal in appearance, more or less marked changes can usually be seen as early as the sixth hour after inoculation. The first change is a slight thickening of the surface epithelium caused by the general edema of the wall of the trachea, enlargement of individual cells, and occasionally multiplication of cells (mitotic figures). Some of the cells may appear practically normal, but the majority of those lining the upper surface lose their cilia and are thrown off and show retrogressive changes, oftentimes the so-called 'balloon degeneration.' Reticular degeneration of epithelial cells in the surface layers is a rare finding. In most of the cases showing a marked edema of the mucous membrane there is a disarrangement of the various parts of the tracheal wall, and in those with a pronounced infiltration the epithelium including the mucous glands is nearly destroyed.

"Formations simulating giant cells occur in the edematous fluid between the surface epithelium and submucosa. By the use of Mayer's mucicarmine stain they can be demonstrated to be mucous glands separated from the surface epithelium and scattered irregularly in the edematous mucous membrane. They are always found compressed by the surrounding edematous fluid and show degenerative changes of the peculiar type described above. Later the nuclei disintegrate, the degenerative products collect in the center, and there is a hyperchromasia and hypereosinophilia. Finally the whole formation may be transformed into a necrotic area in which mucin

can still be demonstrated with specific stains. Fat may be present in large amounts.

"The individual variation in the findings is great, depending upon the length and severity of the process. Secondarily invading bacteria may lead to a partial or diffuse necrosis of the tracheal wall and complicate the picture. Cocci and more frequently diphtheroids, probably similar to those described by Graham and his coworkers, are present in such cases. In general, however, such bacteria seem to play a minor role. Rickettsias have not been found.

"*Tracheal Exudate.*—The composition of the exudate in the trachea differs widely in individual cases. In the early stages there is only a slight proliferation of the upper layers of epithelial cells which are thrown off either singly or in layers and collect on the surface of the mucous membranes, while in other cases there is a mixture of epithelial cells, mucus, round cells, eosinophilic leucocytes, and red blood corpuscles. Sometimes the cellular elements predominate, sometimes the mucus. There are cases in which the mucous membrane is covered with a thin layer of red blood corpuscles. In other cases what appears to be exudate is really edematous mucous membrane which has become separated from the underlying tissue and thrown off in large masses. It is a striking fact that fibrin could seldom be demonstrated in the exudate. In a number of cases the exudate consists of degenerated cells, and sometimes real necrosis is seen. In instances with marked infiltration of the mucous membrane exudate may be entirely lacking.

"*Lungs and Bronchi.*—The changes in these organs are usually restricted to the bronchi and the tubules. The bronchitis, which is almost always present, varies in degree and is often associated with a peri- and parabronchitis. The lesions resemble those in the larynx and trachea but very seldom are as marked. The bronchial exudate is largely made up of the same elements and shows the same variations as that in the trachea. Sometimes bronchiectasis is present. In addition to these lesions, in almost all cases there occurs marked congestion indicated by an extreme filling of the capillary network of the lungs and a more or less pronounced edema. Scattered around the hemorrhages in the bronchial wall and in the congested areas are groups of hemin crystals either in the tissue or included in various cellular elements.

"These lesions in the lungs and bronchi seem to be more extensive in the spontaneous disease than when it is artificially produced. In a few cases a true pneumonia is present in the tissue surrounding

the affected bronchi. In addition more profuse hemorrhages and larger foci of necrosis are found in the lung parenchyma, often walled off by fibroblasts. In many cases numerous foreign body giant cells are found among the fibroblasts."

For comparison I will quote the autopsy findings on a case of acute laryngotracheobronchitis which we had in the Babies' Hospital in 1937. The gross examination revealed the following:

"*Lungs:* On the section the lung surface is a homogeneous red. The walls of the bronchi are markedly thickened. The smaller bronchi are thus made more prominent than usual. Dissection of the larger bronchi reveals thickened grayish walls and occassional masses of thick, tenacious exudate.

"The vocal cords are very edematous, so that there is very little space between them. Their surfaces are granular. The tracheotomy opening is 1 cm. long and extends from 2.5 cm. below the vocal cords to 3 cm. above the bifurcation of the trachea. The tracheotomy wound is lined with greyish granulation tissue, and there is slight infection of the surrounding tissue. The surrounding tissues are separated from the trachea for a distance of 3 to 4 mm. from the margin of the wound. The mucous membrane of the trachea is edematous, granular, and greyish. In the trachea above the level of the tracheotomy wound is thick, tenacious exudate similar to that seen in the bronchi."

Microscopic examination revealed:

"*Lung:* A. Throughout the section there is marked edema, so that many of the alveoli are filled with fluid. There are several small hemorrhages, and there is congestion of the blood vessels throughout. A few areas are emphysematous. The exudate in the alveoli consists mainly of edema fluid, but in a few places lymphocytes, polymorphs, red cells and fibrin are present. The cellular exudate is most marked near the bronchi. All the small bronchi are filled with an exudate of lymphocytes, polymorphs, red cells and fibrin, and in one bronchus the mucosa is eroded. The bronchioles are fairly normal.

"B. In this section there is emphysema with rupture of many of the alveolar walls. There is a small patch of atelectasis, and in a few small areas there is a cellular exudate. Edema and hemorrhages are present but less in extent than in section A. The lymphatics are dilated. The walls of the bronchi are edematous and infiltrated with polymorphs and mononuclears.

"C. In this section the edema and hemorrhages are more marked than in the other sections, but there is little cellular exudate. Throughout large areas the alveolar walls are very indistinct and contain pyknotic and fragmented nuclei. A few clumps of bacteria are seen. A few small areas of the section are fairly normal except for atelectasis.

"*Gram Stain:* Gram positive cocci in short chains and groups of two can be found throughout the section with the exception of the small atelectatic areas. There is very little cellular reaction around many of the clumps of cocci. The cocci are apparently extracellular.

"*Trachea:* A margin of the tracheotomy wound is included. The epithelium is necrotic. The submucosal tissues contain a marked increase of fibrous tissue and many plasma cells. There is edema throughout. Between the trachea and surrounding muscles is a necrotic area which is edematous and infiltrated with polymorphs and mononuclear cells. In the striated muscles surrounding the trachea there is a fibrinous exudate containing polymorphs and bacteria; an adjacent lymph node included in the section is edematous and congested. In the sinuses are many mononuclear cells and a few polymorphs.

"*Gram Stain:* There are many gram positive cocci in the superficial layers of the epithelium and a few scattered in the exudate in the deeper tissues.

"*Larynx:* The epithelium is denuded over most of the surface. Beneath the epithelium throughout the section there is marked edema and an exudation of polymorphs, lymphocytes, red cells and fibrin. There are marked congestion of the blood vessels and a few hemorrhages. There is a thrombus in one small artery."

It can be seen from the sections of both the chicken and the human that there is a marked similarity in the response to infection in the larynx and trachea. Both of the sections show a marked infiltration with mononuclear cells. This seems, on the face of it, to be rather unusual in an acute infection from the hemolytic streptococcus, the pneumococcus and the staphylococcus, but it is characteristic of the tissue response to a virus disease. The tracheal exudate in both groups shows the same type of degenerative cellular disease. Dr. Beach in his study of the immunology of the poultry disease has proved beyond the shadow of a doubt that this condition is due to a filterable substance which has all the characteristics of a virus.

It is for these reasons that I would like to suggest the possibility of the acute fulminating laryngotracheobronchitis as being the result of a virus infection with secondary invasions of the various bacteria which we have found.

The veterinary surgeons in seeking a method of treatment of this disease which ruins large flocks of chickens have discovered that a vaccine can be made from the tracheal exudate after filtration. A measured amount of this material may be applied to the cloaca of young chickens, and in two or three days if a reaction which is characterized by simple redness and swelling appears the chickens seem to become immune to the disease.

Thus far we have not used any vaccine or sera in the humans, but it would seem feasible to concentrate on this problem now rather than just the mechanical methods now employed. I feel very strongly that although statistically chemotherapy seemed to have very little effect on the mortality in the acute disease it should be continued and supplemented by the use of vaccines or transfusions from patients who have recovered from the disease.

580 PARK AVE.

BIBLIOGRAPHY

1. Baum, H. L.: Acute Laryngotracheobronchitis. J. A. M. A., p. 1097 (Oct.), 1928.

2. McNab, J. C.: Acute Streptococcal Infection of the Trachea in an Infant Aged 15 Months. Jour. Laryng., Rhin. and Oto., 30:336, 1915.

3. Baum, H. L.: Some Endoscopic Observations on Laryngo-Tracheo-Bronchitis. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 33:782 (Sept.), 1924.

4. Gittens, T. R.: Laryngitis and Tracheobronchitis in Children: Special Reference to Non-Diphtheritic Infections. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:422 (June), 1932.

5. Hyde, C. J., and Ruckman, J.: Acute Infectious Edematous Laryngitis in Which Recovery Followed Tracheotomy. Arch. Pediatrics, 48:124 (Feb.), 1931.

6. Brennemann, J., Clifton, W. M., Frank, A., and Holinger, P.: Acute Laryngotracheobronchitis. Am. Jour. of Dis. of Child. (April), 1938.

7. Johnson, Malcolm C.: Acute Laryngotracheobronchitis in Infants. Arch. Otolaryng., 17:230, 1933.

8. Kelly, Herman E.: Tracheotomy for the Relief of Laryngeal Obstruction in Children. Arch. Otolaryng., 22:317 (Sept.), 1935.

9. Jackson, Chevalier, and Jackson, C. L.: Acute Laryngotracheobronchitis. J. A. M. A., 107:929 (Sept.), 1936.

10. Richards, Lyman: A Further Study of the Pathology of Acute Laryngotracheobronchitis in Children. Trans. Am. Laryn., Rhin. and Otol. Soc., 1938.

11. Smith, W. J.: Acute Laryngotracheitis in Children. Arch. Otolaryng., 23:420 (April), 1936.

12. Simpson, W. L.: Pneumothorax Complicating Tracheotomy in Fulminating Laryngotracheobronchitis: Report of a Case. Arch. Otolaryng., 26:411 (Oct.), 1937.

13. Beach, J. R.: Bacteriological Study of Infectious Laryngotracheitis in Chickens. Jour. Exp. Med., 54:801, 1931.

14. Seifried, Oskar: Histopathology of Infectious Laryngotracheitis in Chickens. Jour. Exp. Med., 54:817, 1931.

15. Burnet, F. M.: Infectious Laryngotracheitis in Chickens: Immunology. Jour. Exp. Med., 63:685, 1936.

16. May, H. G., and Tittsler, R. P.: Journal Amer. Veterinary Asso., 67:229, 1925.

17. Diseases and Parasites of Poultry. U. S. Dept. of Agriculture, Farmers Bulletin 1652.

XCV

SOME OBSERVATIONS ON THE CONTROL OF TEMPERATURE AND HUMIDITY IN OXYGEN TENTS*

F. W. Davison, M.D.

Danville

I became interested in this problem while trying to determine what are the ideal climatic conditions for the treatment of children who have acute laryngotracheobronchitis. All authors agree that very high humidity is desirable in order to keep the tracheobronchial secretions as fluid as possible, but little has been said about the optimum temperature. The problem is one essentially of air conditioning, not for normal individuals, but for patients who require special climatic conditions. These thoughts naturally led me to literature on the subject of air conditioning. In articles by Yaglou,[1, 2] McCord and Witheridge[3] I found much of the information I needed. Briefly then, the subject I wish to discuss is air-conditioning of an oxygen tent canopy. This problem has greater significance than is implied by my reference to one disease. I suspect that the therapy of many diseases could be improved by affording the patient certain combinations of temperature, humidity and airflow. A working knowledge of the principles of air-conditioning will enable a physician to prescribe any combination of climatic conditions for a given patient, and the prescription can be filled if there is an oxygen tent with a humidifier available. The principles, of course, are those which apply to air-conditioning of a room or building. For the treatment of youngsters having acute laryngotracheobronchitis, we used an oxygen tent canopy rather than an entire room for several reasons. If the air in a room is humidified to 90 or 95 per cent, the walls drip with moisture, and the furnishings and paint are apt to be ruined by excessive moisture. Furthermore, occasionally it is desirable to administer oxygen to these patients, and this can be

*From the Department of Otolaryngology and Bronchoscopy, The George F. Geisinger Memorial Hospital, Danville, Pennsylvania. I wish to acknowledge the assistance of William Pratt, M.D., in making oxygen determinations and other observations.

Read before the twenty-third annual meeting of the American Broncho-Esophagological Association, New York, N. Y., June 5, 1940.

Fig. 1. Mechanical humidifier with inflow and outflow pipes to connect with an oxygen tent canopy.

most easily done if the tent canopy is already in place over the patient. More accurate temperature control is another advantage provided by a tent. To obtain high humidity without high temperatures we used the home-made modification of the mechanical humidifier* shown in Fig. 1. The intake and outflow pipes are flexible metal tubing of the type sometimes used for exhaust pipes on automobiles. They are introduced into the tent through tight fitting sleeves and thus permit recirculation and humidification of the tent air without loss of oxygen.

Small children and sick adults cannot analyze and describe their sources of discomfort. Therefore, when using a tent we should supply climatic conditions which we know will be comfortable. The temperature and humidity within the canopy have more to do with the comfort of the patient than do the oxygen and carbon dioxide concentrations.

The following sentences are quotations from the 1940 Heating, Ventilating, Air-Conditioning Guide. "Combinations of temperature, humidity and air movement which induce the same feeling of warmth are called thermo-equivalent conditions. A series of tests at the A.S.H.V.E. Research Laboratory in Pittsburgh established the equivalent conditions met with in general air-conditioning work. This scale of thermo-equivalent conditions not only indicates the sensation of warmth but also determines the physiological effects on

*Manufactured by the A. C. Gilbert Co., New Haven, Conn.

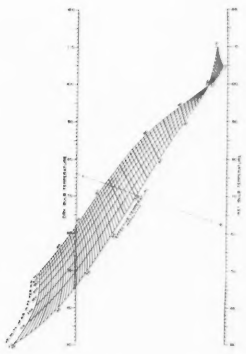


Fig. 2. Effective Temperature Chart. (Reprinted by permission from A.S.H.V.E. Research Report 717, Transactions, Vol. 41, 1935.)

the body induced by heat or cold. For this reason, it is called the effective temperature scale or index. Effective temperature is an empirically determined index of the degree of warmth perceived on exposure to different combinations of temperature, humidity and air movement. It was determined by trained subjects who compared the relative warmth of various air conditions in two adjoining conditioned rooms by passing back and forth from one room to the other."

"Any air condition has an effective temperature index of 60 degrees, for instance, when it induces a sensation of warmth like that experienced in calm air at 60 degrees saturated with moisture. The effective temperature index cannot be measured directly, but it is computed from the dry and wet bulb temperatures and the velocity of the air."[4]

The chart, Fig. 2, shows how effective temperature index can be quickly determined from the wet and dry bulb temperatures if air velocity is known.

Some pertinent data are given by the comfort chart shown in Fig. 3. This indicates that during the winter an effective temperature index of 66 degrees produced comfort for 97 per cent of the subjects tested, and that during the summer an effective temperature of 71 degrees was comfortable for 98 per cent. This suggests the air conditions in which the body is able to maintain thermal equili-

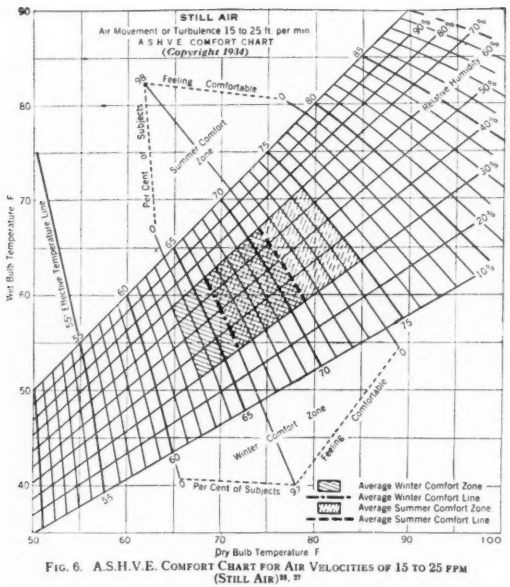


FIG. 6. A.S.H.V.E. COMFORT CHART FOR AIR VELOCITIES OF 15 TO 25 FPM (STILL AIR)[26, 27]

Fig. 3. A.S.H.V.E. Comfort Chart. (Reprinted by permission from A.S.H.V.E. Transactions, Vol. 38, 1932.)

brium with its environment with the least conscious sensation to the individual and with the minimum physiologic demand on the heat-regulating mechanism.

It should be realized that many of the dial type instruments calibrated for the measurement of relative humidity are inaccurate and register very slowly. Wet and dry bulb thermometers are the most accurate means of determining relative humidity, but a fact lost sight of by many people who use them is the necessity for providing adequate airflow past the thermometer bulbs for several minutes before a reading is taken. The following sentence is found on the relative humidity tables furnished with wet and dry bulb thermometers: "These values are correct only for an air velocity of not less than 600 feet per minute." Unless this rate of air velocity is provided, relative humidity calculations will be anywhere from 10 to 25 per cent too high. The drier the air, the greater the percentage of error. We have found the psychrometer with motor-

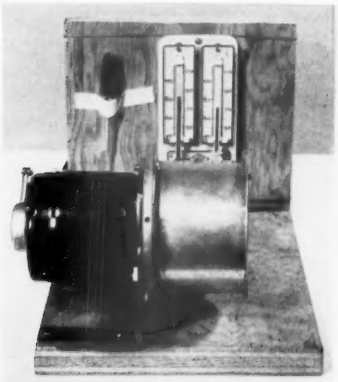


Fig. 4. Psychrometer with motor-driven fan for rapid accurate determination of relative humidity.

driven fan illustrated in Fig. 4 a very convenient and satisfactory method of producing sufficient airflow to obtain an accurate relative humidity determination after two minutes' running. It is small enough to be easily introduced under the tent canopy.

The psychrometer with motor-driven fan is necessary only for experimental work. We think that all tent canopies should be supplied with a wet and dry bulb thermometer hanging free, several inches from the side of the canopy so that it will register fairly accurately the condition of the air circulating within it. When the relative humidity was approximately 50 per cent, the determinations made without the motor-driven fan were only 8 per cent too high. When the humidity was above 70 per cent, the determinations were the same whether the fan was turned on or off.

The control of temperature within the tent canopy is fairly simple, provided the cooling unit has adequate capacity. The damper controlling the volume of airflow through the cooling chamber can be opened or closed just the right amount to achieve the temperature desired within the canopy. Some oxygen tents have a variable speed motor, and this permits regulation of the rate of airflow through the cooling chamber, so in this way the temperature of the canopy can also be controlled.

The motor-blower of the tent we were using puts 100 cubic feet of air per minute at its top speed. Theoretically, then, the air in the canopy whose content is -5 cubic feet, passes through the ice chest about six times per minute. This produces rather rapid airflow according to ordinary standards of air-conditioning, but this is necessary to produce comfortably low temperatures when the room air is hot. In order to avoid an uncomfortable draft on the patient, the air deflector at the outlet of the ice chest should be turned upward. An air velocity of 50 feet per minute is the maximum usually recommended for comfort.

A feature stressed by manufacturers of oxygen tents is their dehumidifying action. We have found the reverse to be true under average winter conditions. For example, on March 26, 1940, the hospital air had a temperature of 80 degrees and a relative humidity of 17 per cent. After the oxygen tent had been running for 60 minutes with the motor at top speed, the air in the tent canopy had a temperature of 60 degrees and a relative humidity of 39 per cent. The relative humidity within the canopy is not difficult to regulate at a desired level anywhere between 40 and 80 per cent by the addition of a humidifier. Humidities higher than 80 per cent are hard to achieve if airflow through the ice-chest is rapid, because atmospheric moisture is quickly precipitated in the ice chamber. The problem here is to add water vapor to the canopy more rapidly than it is removed by the cooling chamber. By using a humidifier which evaporates one pint of moisture per hour. we have been able to keep the relative humidity at 95 per cent with a temperature of 70 degrees. This produced an effective temperature of 60 degrees, which is entirely comfortable. If a steam generator is connected to the canopy, conditions such as are present in the average so-called croup tent are produced, namely, temperature of 90 degrees and relative humidity of 95 per cent; that is, an effective temperature index of 80 degrees. This is an exceedingly oppressive and uncomfortable atmosphere, as I found out by personal experience, yet for generations we have been subjecting croupy youngsters to these conditions with little thought of the discomfort produced.

A relative humidity lower than 40 per cent is difficult to achieve with an ice cooled tent because the film of moisture on the melting ice adds water if the air circulated past it is already dry. Lower humidities can be achieved by use of a tent cooled by mechanical refrigeration by solidified carbon dioxide or by circulating the air as it leaves the cooling chamber over beds of silica gel. We have not yet experimented with these methods.

From a theoretical point of view, we felt that adding oxygen might reduce the humidity, but such was not the case. A flow-rate of eight liters per minute did not reduce the relative humidity a measurable amount but would maintain the oxygen concentration at 50 per cent.

Any discussion of this type brings us naturally to the consideration of what we are doing to the physiology of our patients when we artifically raise or lower either temperature or humidity from the average conditions to which they are accustomed. Ordinarily we think of an oxygen tent as a means of raising the oxygen content of inspired air. It may also be thought of as a means of controlling heat loss and water loss from the body. When properly regulated, a tent canopy is a useful means of controlling these two highly important functions. Heat loss from the body may be considered under two headings: sensible and latent. Sensible heat loss takes place by radiation and conduction.[5] It depends largely on dry bulb temperature and stops when the temperature of the air reaches that of the body. It is not surprising that children are restless, uncomfortable and irritable in the old-fashioned type of croup tents wherein the temperature was raised to 90 or more degrees in an effort to achieve high humidity. Latent heat loss occurs by evaporation of perspiration and respiratory moisture. It is controlled by the temperature regulating mechanism of the body and is affected by air motion and humidity of the atmosphere. It is again evident that the lack of air motion in an old-fashioned croup tent must be a source of considerable discomfort. We should not lose sight of the fact that perspiration induced by raising the temperature and humidity can cause excessive water loss. Conversely, raising the humidity while keeping the temperature low enough to avoid perspiration is a means of conserving the body's supply of water. Evaporation of respiratory moisture is decreased if humidity is high. Thus, maintaining a high humidity with the temperature not above 70 degrees can be a useful method of conserving body fluids so that an adequate volume will be available for kidney function.

CONCLUSIONS

1. The temperature and humidity of an oxygen tent have more effect on the comfort of the average patient than does the oxygen concentration.

2. A wet and dry bulb thermometer should be part of the equipment of every oxygen tent.

3. Temperatures of 68 and 70 degrees are usually the most comfortable when humidity is high.

4. Humidity in a tent can be raised to 95 per cent when the temperature is 70 degrees by the use of a mechanical humidifier.

5. An ice cooled oxygen tent with a motor is a useful, practical means of increasing heat loss from the body.

6. Water loss from the body can be decreased by increasing the humidity if the air temperature is not above 70 degrees.

GEISINGER HOSPITAL.

BIBLIOGRAPHY

1. Yaglou, C. P.: Physical and Physiologic Principles of Air-Conditioning. J. A. M. A., 108:1708 (May 15), 1937.

2. Yaglou, C. P.: Physical and Physiologic Principles of Air-Conditioning. J. A. M. A., 109:945 (Sept. 18), 1937.

3. McCord, C. P., and Witheridge, W. N.: The Measurement of Air Conditions. J. A. M. A., 111:1647 (Oct. 29), 1938.

4. Heating, Ventilating, Air-Conditioning Guide, 18:57, 1940. Published by the American Society of Heating and Ventilating Engineers, New York.

5. Wright, S.: Applied Physiology. Oxford University Press, sixth edition, New York, p. 522, 1938.

XCVI

BRONCHIAL COLLAPSE WITHOUT CLINICAL SIGNS OF EXTERNAL PRESSURE: BRONCHOSCOPIC OBSERVATIONS*

HERRMANN E. BOZER, M.D.

BUFFALO

The bronchoscopic observation of the collapse of a main bronchus, without any evidence of external pressure, is probably rather uncommon. A fairly careful search of the literature for the past twenty years failed to reveal any paper concerned with such observations. It was, therefore, thought advisable to call attention to this condition in the hope that other such cases may be recognized and studied with the intent of definitely establishing the true etiologic factor concerned in this condition.

This report covers the experience of three such cases, two of which ended fatally. Unfortunately, autopsy was not permitted in either case. Thus any discussion concerning the mechanism of the collapse must be to a great extent purely speculative.

CASE 1.—J. D., 19 years old, entered the Marine Hospital of Buffalo, September 18, 1931. His chief complaint was shortness of breath.

Past History: This was negative except for measles during childhood. He denied venereal disease. During the past year he had passed a physical examination for a position in the United States Coast Guard.

Present Illness: On September 13, 1931, while riding in an automobile, he was taken with a sudden mild shortness of breath and a mild cough. He thought he was taking a cold and did nothing about it until two days later. Then he consulted a doctor, who diagnosed a coryza and chest cold and gave the patient a cough syrup. The dyspnea and cough increased, and the patient entered the Marine Hospital three days later.

---

*Read before the twenty-third annual meeting of the American Broncho-Esophagological Association, New York, N. Y., June 5, 1940.

Examination at this time showed a well-nourished young man of average build, apparently not acutely ill except for a moderate degree of dyspnea. Temperature was 99.8 degrees, pulse 100, and respiration 24. Physical examination gave the following positive findings: the right side of the chest was more prominent than the left; there were no fremitus or breath sounds on the left side; the heart was displaced to the left; the sounds were of poor quality; there was moderate degree of scoliosis of the back; the urine was negative.

An x-ray taken September 22 showed a massive atelectasis of the left lung. During the patient's six-day stay in the hospital, his temperature varied from normal to 100.5 degrees, being irregular in type. The pulse gradually increased to 120 and the respiration to 30 per minute. The cough increased and there was considerable pain at times in the left chest.

After consultation, bronchoscopy was advised. The patient, accompanied by a medical officer, came to the Buffalo City Hospital to have the examination made. We first saw the patient on his arrival there, and in no way did he appear dangerously ill.

Operation: The patient was anesthetized in the customary manner for bronchoscopy, 10 per cent cocaine being used. A seven millimeter bronchoscope passed easily into the trachea down to the bifurcation. It was then observed that the left bronchus was completely collapsed and displaced upward and to the right of its usual position. The right bronchus was of normal shape and appearance. There was no inflammatory reaction of the membranes of the trachea or right bronchus. The bronchoscope at no time was passed into the right bronchus. A very small, flexible, filiform bougie was carefully inserted into the left bronchus. This was followed by a side-curved forcep which was gently expanded. Then for the first time, breath sounds could be heard in the left lung through a stethoscope. After holding the bronchus open for approximately two minutes, it was found to remain about half open upon withdrawal of the forcep. The orifice of the bronchus also drew over more to the left and downward although not yet in its normal position. The membranes of the bronchus were not reddened or inflammatory in appearance, and there was no pus. The bronchus wall was not thickened. As the patient had now secured partial use of the affected lung, we felt that it would go on to complete expansion of its own accord. The bronchoscope was withdrawn. The patient sat up on the table, smiled and seemed relieved. As he started to speak, his whole ap-

pearance suddenly changed. A look of extreme surprise and fear came over his face and he immediately became extremely dyspneic and cyanotic. As he was struggling for air, it was impossible to repass the bronchoscope. It was also apparent that his obstruction was not laryngeal. Auscultation of the chest showed no breath sounds on the right side—the previously good side—and impaired breath sounds on the left, that which previously had had none. Oxygen was immediately supplied, and general measures to combat shock and failing heart—all to no avail. The patient died in about ten minutes. Autopsy was refused, but a postmortem x-ray showed pneumothorax of the right side, the lung being collapsed down into a very small mass. The left side still showed no effects of the complete atelectasis—that air which had passed into the affected lung not having had time to influence the x-ray picture.

Comment: The onset of this patient's illness was sudden. The feeling of a "tightness" in the left chest was present from the first and was soon followed by dyspnea. It would seem that the bronchial collapse probably started from the first, and became complete in a few days. It may be that a slight upper respiratory infection did precede the bronchial collapse, but even so, cough was not a marked symptom at first. And if it was the exciting factor in the collapse, certainly the bronchial structure must have been very weak not to have withstood this slight strain, and it is surprising that the collapse had never occurred before.

The fact that the bronchial wall was able to maintain a partially open position when expanded by forceps would indicate at least the presence of some degree of structural function. However, in view of the fact that expanding or stretching of spastic points of a hollow viscus overcomes the spastic effect, e.g., cardiospasm, the possibility of a strong spasm of the bronchial muscle cannot be entirely discarded. The unfortunate outcome was in no way the result of the collapsed bronchus, except that if the right lung had been functioning normally, the patient would probably not have succumbed to the pneumothorax of the opposite side. The pneumothorax undoubtedly was due to the rupture of a posterior bleb caused by the emphysema of the right lung, a normal condition when one lung has to do the work of two, as was the case with this patient for several days.

CASE 2.—F. B., aged two months, colored, attended the Out-Patients' Department of the Buffalo Children's Hospital, September 2, 1932. The chief complaint was difficult breathing.

Family History: The father, aged 24, and the mother, aged 21, were alive and well. There was no history of tuberculosis, lues or malignancy. There had been no miscarriages or abortions.

Past History: The child was full term and delivered normally. There was no cyanosis, nor convulsions. The birth weight was seven pounds. It was breast fed and was getting eight cc. of orange juice daily. It had been well until the onset of the present illness.

Present History: For the past two weeks the mother had noticed that the child breathed faster than previously and that its chest was covered with perspiration. She also noticed that it would become quite cyanotic when it cried, and sometimes when not crying.

Examination revealed the following findings: Well-developed and well-nourished baby, two months of age, weighing ten pounds; temperature 99 degrees; apparently not acutely ill; head—normal in shape, fontanelles open; pupils of eyes equal and reacted to light; ears normal; nose—no obstruction, no discharge; throat—mucous membrane clear, not injected; no adenopathy or rigidity of the neck.

Chest: expansion decreased on the right side; left side shows some evidence of emphysema and the ribs are farther apart than on the right side. Lungs: decreased resonance on the right side; normal breath sounds on the left; no râles. Heart: pushed to midline; apex beat heard at fifth interspace, 2 cc. from mid-sternal line; regular rate rhythm; no murmurs.

Abdomen: normal; no masses; organs palpable; no muscle rigidity or tenderness.

Genito-urinary: extremities, reflexes, and skin normal.

Impression: atelectasis of upper lobe, right side.

Recommendation: x-ray and see again.

An x-ray, September 2, 1938, showed atelectasis, upper lobe right lung.

On September 9, 1938, the mother brought the child to the hospital where he was admitted to a ward. She related that during the past week the dyspnea had increased considerably. Just before coming to the hospital the respiration had been extremely labored and the child had had a "spasm".

Examination proved the same as previously noted and the diagnosis was the same. Temperature 99 degrees; pulse, 140; respirations 60(?).

The child's dyspnea gradually grew worse, and I was called to see the patient about 3:00 p. m. the day after admission. At this time, the infant was in a critical condition and near asphyxia. Bronchoscopic examination was proposed but before this could be carried out, the dyspnea increased to such a dangerous state that a quick emergency tracheotomy was performed and a long rubber catheter was introduced into the trachea with suction, followed by direct insufflation of oxygen. Although apparently no mucus or pus was aspirated, the child improved with this treatment and bronchoscopy was deferred until the infant should be in a better condition. Just previous to the operation, the same clinical signs prevailed as on previous occasions, i.e., suppressed breath sounds of the right side and clear breath sounds and resonance on the left.

The child was somewhat improved for a few hours, but about five hours after the first operation I was again called. This time also he was in an exceedingly bad condition due to dyspnea. However, at this time, the physical findings were reversed: the suppression of breath sounds were on the left side and normal breathing could be heard on the right side. The temperature was 100 degrees; pulse, 60; respiration, 55 (?).

Bronchoscopy was performed without difficulty, a four millimeter tube being used. There was no evidence of inflammation or pus in the trachea. The carina was sharp and in its normal vertical position. The right bronchus appeared absolutely normal. The orifice of the left rear bronchus was closed. A small side-curved forcep was introduced into the left bronchus and gently opened. The bronchial wall was flaccid, and the collapse extended for an estimated distance of about one centimeter. As long as the bronchus was held open by the expanded forcep, breath sounds were clearly heard over the left lung by stethoscope. Upon closure of the forcep, the bronchial wall would immediately collapse and no breath sounds could be detected. This maneuvre was repeated several times, always with the same results. The physician listening at the stethoscope reported that the air would enter with a rush and shut off just as rapidly, coincident with the manipulation of the expanding forcep. Unlike the previously reported case, the bronchial walls showed no ability to regain a normal position. No abnormal secretion was visualized in the bronchus while it was kept expanded. After the examination the child was again returned to an oxygen tent. The temperature never ran higher than 100.6 degrees, while the pulse gradually increased from 140 at the time of the operation to a rate that was no longer countable, and the respiration held to approximately 60 per

minute. The child died eight hours after the operation. Autopsy was refused.

Comment: This case, from the first, presented the history and clinical evidence of bronchial obstruction of the right side. Whether this was due to a mucus plug or a collapse of the bronchus, it is impossible to say as the critical condition of the child demanded tracheotomy when first seen, and the bronchial obstruction was released at once by the insertion of the rubber catheter with suction. Although one might readily assume that a mucus plug was aspirated, nevertheless, the mere passing of a rather stiff catheter might have opened up the bronchus.

The collapse of the left bronchus, however, was seen, and was sudden and complete. Here again we may assume, if we so wish, that the strain of the severe coughing following tracheotomy and aspiration may have weakened a structurally weak bronchus and caused its collapse. The very flaccidity of the bronchial walls as demonstrated through the bronchoscope by the expanding forceps would point to this conclusion. The advisability of having various sizes of bronchial intubation tubes at hand is demonstrated in this case.

CASE 3.—Mrs. V. B., aged 40, entered the Buffalo City Hospital, March 22, 1937. She gave the following history: chief complaint—severe paroxysms of coughing, followed by marked dyspnea; past history—negative except for frequent headaches and several attacks of tonsillitis.

The present illness had begun seven years previously. Following rather severe exertion (shovelling snow), the patient suddenly developed a paroxysm of coughing. This was very severe and left her quite dyspneic. She had no upper respiratory infection at this time. Following this attack she developed others, always after severe exertion. Two years ago, the attacks became more frequent and would come on after any, even mild, exertion. The dyspnea at this time could be relieved only by rest and adrenalin. For the past two years she had been able to do very little work. The dyspnea never came on without a preceding paroxysm of coughing.

General examination gave practically negative findings except for a few bronchial wheezes in both lungs, and a suppression of breath sounds anteriorly on the left side. She also had a pair of chronically infected tonsils. X-ray of the sinuses showed a slight cloudiness of the left ethmoid. X-ray of the chest showed at the

right side, an area of suspicious infiltration of the third interspace anteriorly. The remainder of the lung field showed only increased lung markings and a suspicion of bronchiectasis in the right base. The diaphragms were normal in position and contour. The heart was slightly enlarged.

The patient was bronchoscoped March 23, 1937. Pontocaine anesthesia was used. The patient was very cooperative, and the examination was easily made.

The trachea was normal, but the carina was deviated at an angle of forty-five degrees to the left. The left bronchus was nearly completely collapsed, extending down from the carina. All the mucous membranes were apparently normal, and there was no abnormal secretion. No manipulation was done.

One week later, bronchoscopy was again performed and the tracheo-bronchial tree was entirely normal. Neither at this examination nor at the first one did the mucous membrane have any of the typical appearance of allergy.

The patient continued having her typical attacks at frequent intervals until several months later. At that time, tonsillectomy was performed, and until the present time she has never had another attack although she now does all her own housework.

Comment: There is no doubt that this patient had allergy producing asthmatic symptoms. Adrenaline always gave relief. Whether the collapse of the bronchus was due to the bronchospasm or a result of the severe paroxysm of coughing it is difficult to say. There is always the possibility that the bronchus collapsed first and that the paroxysmal cough was due to this condition. However, it would seem more probable that the collapse was due to the strain put on the bronchial structure at the time of the severe cough. That this strain is considerable can readily be observed bronchoscopically during severe coughing, when the bronchial walls are often forced nearly into apposition from the great intrathoracic pressure.

The structure of the main bronchus is substantially that of the trachea. The framework consists of the so-called rings of hyaline cartilage which form some three-fourths of a circle. These rings are about two to three millimeters in breadth and form the anterior and lateral supports of the bronchus. The distance between the rings is generally less than their breadth. The length of the left bronchus to its first branch is about 5 cm. and contains eight to ten rings. The right bronchus is generally about 2 cm. long and contains from

two to four rings. A highly elastic fibrous sheath, continuous with the periosteum of the cartilaginous rings, complete the posterior portion of the bronchus.

In the main bronchus, the bronchial muscle forms a thin layer connecting the ends of the cartilaginous plates. In addition there are some oblique and longitudinal fibers.

A layer of connective tissue forms a submucous layer, separating the muscle layer from the mucous lining. This layer contains some fat cells and many tracheal glands.

The mucous layer is formed of stratified, ciliated, columnar epithelium which contains numerous secreting goblet cells.

The inervation of the lungs comes from two sources. Afferent and efferent fibers come from both the spinal sympathetic system and the vagus system. The spinal sympathetic fibers pass by way of the rami communicantes albae, stellate ganglion, ansa subclavia, and through the vagus nerves to the lungs of the same and opposite side. These fibers function as vasomotors. The vagus efferent fibers rise from the dorsal vagus nuclei and reach the lung by the vagus nerves.

From the anatomy of the bronchus, it can thus be seen that stimulation of the vagus nerve peripheral to the ansa subclavia should effect all the above-named fibers to the lung. Dr. Byron Francis has performed this experiment and caused a definite narrowing of the bronchial lumen. However, it would seem unlikely that without a severe structural weakness of the bronchial wall, a complete closure of the bronchus could be so affected. That such structural defects are possible has been shown by Van Czxlards,[4] who believed he demonstrated a congenital weakness of the elastic components; and Trump, who in an autopsy of an eighteen-months-old girl, demonstrated that all the cartilaginous rings were missing except for a small plate articulating normally with the cricoid and the upper two tracheal rings.

It may be significant that in all three cases described here, the left bronchus alone was involved. This bronchus is longer than the right and should therefore be more susceptible to collapse. This coincides with our observations made during the bronchoscopic examinations of cases of asthmatic bronchitis, that the left bronchus always is more compressed by the severe cough than the right. The very flaccidity of the bronchial wall in Case 2 would point to structural weakness rather than spasm of the bronchial muscle. However, in Case 1, it would appear difficult to believe that a bronchus could

collapse completely without a greater exciting cough than the patient stated he suffered. Without the definite information of the bronchial structure in these specific cases, which an autopsy would have revealed, it is impossible to draw any certain conclusions, but it would seem not improbable that failure of the normal structure of the bronchus, with or without an accompanying bronchospasm was able to cause complete collapse of the bronchus, resulting in atelectasis. It may well be that more of these cases occur and are not diagnosed due to lack of bronchoscopic examination.

The above three described cases illustrate the results of collapse of a main bronchus. In two of the cases, the obstruction was complete. The diagnosis was established by bronchoscopic vision and so the nature of the obstruction is unquestionable. No infiltration of the bronchial wall or benign tumor was present, nor were there foreign bodies or mucus plugs. X-ray studies practically eliminated the possibility of the collapse being due to pressure from enlarged mediastinal glands, tumor, or aneurysm. It would, therefore, seem entirely fair to assume that the cause of the collapse was intrinsic in the walls of the bronchus itself. Following this assumption, the only two methods of collapse that present themselves are, first, the occurrence of an exceedingly strong spasm of the bronchial musculature; second, the presence of structural weakness in the wall of the bronchus.

468 DELAWARE AVE.

BIBLIOGRAPHY

1. Macklin, Charles C.: Functional Aspects of Bronchial Muscle and Elastic Tissue. Archives of Surgery, 19:1212, Part II (Dec.), 1929.

2. Larsell, O.: The Ganglia, Plexuses and Nerve Termination of the Mammilian Lung and Pleura Pulmonalis. The Journal of Comparative Neurology, Vol. 35, No. 1 (Dec.), 1922.

3. Francis, Byron: Changes in the Shape and Size of the Tracheo-Bronchial Tree Following Stimulation of the Vago-Sympathetic Nerve. Arch. of Surgery, 19:1577, Part II (Dec.), 1929.

4. Hencke, Lubarsch, Von Hans, Joachim, Arndt und Anderen: Atmungswege und Lungen, Bearbeiet. Handbuch der Speziellen Pathologischen, Anatomie und Histologie, Band III, Teil 3, 1931.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, February 5, 1940

THE PRESIDENT, DR. GEORGE T. JORDAN, IN THE CHAIR

Symposium on Syphilis:
Interpretation of Serologic Reactions Based on a Nation-wide Survey

FRANCIS E. SENEAR, M.D.

(Abstract)

The interest in the serology of syphilis has perhaps not kept pace with the interest in the more general phases of the subject, so far as the profession at large is concerned, and particularly with respect to the public. However, the field has become just as complex as that of the therapy of syphilis.

Since 1923 three meetings have been held under the direction of the League of Nations for the purpose of making a study of the various tests used in the diagnosis of syphilis. At the third meeting held in Uraguay in 1938, it was concluded that the Kahn test was the best. The American Society of Clinical Pathology, however, recognizing that a number of tests developed in the United States had not been adequately represented, asked Surgeon-General Parrin to arrange a further study as a result of which a call was sent out to thirteen outstanding serologists who had developed tests or modified already existing tests so that they were considered separate tests. Blood was sent to these men and examined in their own laboratories, while in the League of Nations' competitions the serologists went to the country in which the contest was held and competed in strange surroundings. Following this first study, five men whose tests were outstanding were chosen as controls for further work of this organization. These men were Kahn, Klein, Kolmer, Hinton and Eagle.

Each of these men, with the exception of Kolmer, uses a flocculation test; Eagle and Klein also have developed complement fixation tests, but they are not used as much as the flocculation test.

It was then decided to find out the general calibre of laboratory work throughout the country. Invitations were sent to a number of laboratories, state, municipal, hospital and private; in the study as it pertained to those using the Kahn test, Kahn performed the control tests, and the same was true with the other tests. It was interesting to find that in the known syphilitic sera assigned to those using the Kolmer test, for example, Kolmer obtained 59 per cent of positive results. This does not mean he should have obtained 100 per cent, because many were treated cases or were of such long standing that the reaction had spontaneously become negative. In 14 laboratories using the Kolmer test, the reports were anywhere from 32 to 62 positive sera reactions, some laboratories obtaining positive reactions in not more than 50 per cent where the reactions should have been positive.

The next study applied to the state laboratories alone. In the first study 39 state laboratories participated. Each received a specimen of blood, and the various control men received exactly comparable samples. Two of the state laboratories reported 8 and 10 per cent of false positive reactions respectively. In this same study the control laboratories reported positive reactions in 87 per cent of the known syphilitic sera, while the general average of state laboratories was 16 per cent below—they failed on the average to detect syphilis in 16 out of 86 cases where it should have been possible to do so.

The next year a similar study was made with 44 state laboratories participating. As a consequence of the previous studies, they had taken steps to improve their technic, and in this study their results were only 9 per cent below the control group. None had over 2 per cent of false reactions. A third study was carried out the following year in which only 38 laboratories participated; 10 whose results had been good were asked not to participate because the collection of blood was such a complicated and laborious test, 250 to 275 cc. of blood being taken from each individual, either syphilitic or control. In this test the number of laboratories classed as unsatisfactory dropped to 22 as compared to 30 in the preceding year.

As to what can be done with good technic in the performance of laboratory diagnosis of syphilis, it is interesting to note that in

the 1938 study there was not a single false positive or false doubtful case reported by any of the control serologists. Next month the annual study for this year will be started, and a steady improvement is expected. This brings up a brief consideration of what should be looked for in the interpretation of reactions, what we should know of the possibilities of diagnosis of syphilis from the laboratory standpoint. In the early stage the reaction is negative; it is ordinarily ten days to two weeks before we get any appreciable portion of positive reactions. In a week one may expect 25 per cent, and with the flocculation test the reaction becomes positive somewhat earlier. Toward the secondary period the proportion of positives becomes greater, and 100 per cent of positive reactions have been found in the secondary period by the control serologists. In the later or tertiary stage, the proportion drops very materially; you will find only about 75 per cent showing positive reactions. In congenital syphilis and paresis the proportion is high, about 96 to 98 per cent. Tabes, on the other hand, runs very low, 65 to 70 per cent. Yaws, or frambesia, a tropical disease which we rarely see in this country, gives a high proportion of positive reactions, about 75 per cent, and leprosy gives 40 to 70 per cent positive reactions. Malaria will give rise to a considerable proportion, particularly in the febrile stage. Various other conditions such as jaundice, malignancy, pregnancy, diabetes, febrile diseases and tuberculosis have been capable of producing biologic false positive reactions, but they are of influence in only a small proportion of cases if at all. Recent studies by the National Tuberculosis Association show that all tuberculosis may be discarded as a cause of positive reaction. Malignancy and other things mentioned are rarely of importance, but in infectious mononucleosis, if the blood is taken at the right time, a positive reaction is often obtained. Children running a febrile course occasionally show positive reactions, the reaction reverting to negative after disappearance of the febrile disease.

There is one type of reaction which should be mentioned, the so-called paradoxical reaction. Patients with no history or signs of syphilis will, in rare instances, show a positive reaction today, usually weak; a week from today it will be negative; and within another two weeks will be weakly positive. The same laboratory one day may report a negative Wassermann and a positive Kahn; or on blood drawn at the same time, one laboratory will report a 2+ positive and the other, a negative reaction. Spanswick estimated that this occurred once in 5,000 tests. These reactions are very difficult for the syphilologist to evaluate, as one does not know whether to treat

the patient or let him go untreated. They are encountered occasionally, and cases of this type usually turn out to be non-syphilitic, biologic false positive reactions.

In summary, one should know the quality of the work done by the laboratory he employs. The standard is undoubtedly being raised and will continue to be raised as the state laboratories extend evaluation opportunities to the laboratories within their confines. A poor laboratory may miss a lot of positives or get false positives many times.

Lesions of the Mouth

JAMES H. MITCHELL, M.D.

(Abstract)

The lesions of the mouth in syphilis may be divided into early, late and congenital. The most important part of early syphilis is the primary lesion, and the most common site for this is the lip, usually the lower. You are all familiar with the appearance and character of that lesion, the chancre. It is not an ulcer but an erosion, a very hard, palpable lesion, a button. It is absolutely without sensation and ordinarily without secondary infection. We have possible sources of error in various other lesions such as a herpetic lesion which has been treated with irritants to the point where it has become indurated and simulates a chancre to a marked degree. However, a most important diagnostic point is the regional adenopathy, which is occasionally bilateral but usually on one side or the other, and painless on palpation.

When the crust is removed from one of these lesions, a very clear serum exudes, and in this serum we find the spirochete pallida by means of the dark field. All lesions about the mouth must be very carefully examined, because we have an organism in the mouth which is so much like the spirochete pallida that I would not attempt to differentiate the two organisms.

The tongue is the next most common location for the chancre, and then the tonsils, in regard to which I regret to say I have seen a great many cases in which tonsillar chancres have been removed. This is not by way of criticism, but I have seen a large number that have been removed by tonsillectomy, and I regret also to say that

I have seen a number of chancres inside the nares of the nose and throat men who have come in contact with chancres and have acquired the lesion through carelessness. I have seen a number of chancres in the eye. The gum is a very rare location for chancre. In the tonsil the chancre is often mistaken for Vincent's angina. If it were a really pronounced case of Vincent's angina, there would be probably some fever and some adenopathy, but not as with a true chancre.

Rhinologic Aspects

LAWRENCE J. LAWSON, M.D.

(Abstract)

Syphilis of the nose is important because a high percentage of those infected with syphilis, unless actively treated, sooner or later develop nasal changes. It can cause serious threat to life. Its sequellae are of serious character. Permanent disfigurement causes social limitations. There is interference with speech and character of voice. Changes occur in respiration and deglutition. Insidious symptoms are often obscure. At times the lesions progress with extraordinary rapidity.

The lesions of hereditary and acquired syphilis, as seen in the nose, were described in detail, and the essayist concluded his presentation by saying that it is fair to presume that factors now at work, if not interrupted by war or lowered economic status, will reduce the incidence of advanced syphilitic nasal damage to a very low level. Certainly this is becoming true in private practice and in the clinics. Modern American medicine is alert and progressive. Blood testing of all pregnant women in Illinois is now required by law. With both prenatal and premarital tests and the growing tendency for group testing leading to early recognition of syphilis, fewer of the more serious manifestations of syphilis of the nose will be allowed to develop.

The high number of fresh infections quoted in current reports makes it important that syphilitic manifestations be recognized, especially those of the nose, where involvements are frequent.

Otological Aspects

SAMUEL J. PEARLMAN, M.D.

(Abstract)

Few otologists see many cases of syphilis of the ear. It may be that a number are missed, but in general it is agreed that syphilis of the ear is not very common.

Chancre and condylomata occur on the concha and in the external auditory canal. Chancre was reported not unfrequently in European clinics and said to be due to the improper sterilization of hard rubber catheters. Stenosis of the eustachian tube can also occur as a sequel to syphilitic scarring of the nasopharynx.

Syphilis of the ear may be divided into three main groups:

1. Late hereditary type: This comes on about puberty, is associated with other stigmata of hereditary syphilis as described by Hutchinson and produces a sudden and incurable deafness of the perception type.

2. Infantile prenatal type: The patient is born often with obvious and severe evidences of syphilis involving skin, viscera, brain, etc., and the death rate is high. Many of these children do not live longer than a few weeks or months.

3. Lastly, in the acquired type, the main findings are deformities of the footplate of the stapes, which may be bowed in or out, and deformities of the crura. Periostitis gummosa may be seen, according to some, in the middle ear, in the region of the ossicles and in the mastoid process. One of the most interesting findings is a productive periositis (non-gummatous). In addition, there are varying degrees of atrophy of the spiral ganglion cells and degenerative changes in the organ of Corti, as well as of the acoustic nerve in Rosenthal's canal and of Reissner's membrane. Round cell infiltration occurs in the cochlea along the basement membrane, in the striae vascularis, in the spinal ligament, the modiolus, and in the internal auditory meatus along the fibres of the eighth nerve.

Some of the round cell infiltration is perivascular.

As for diagnosis, there are few instances where the symptoms are unique for syphilis. The late congenital types occur at or about puberty and are characterized by Hutchinsonian stigmata, sudden

onset of the deafness, which is profound, and labyrinthine phenomena.

There are some who claim that the findings in syphilis are characteristic along with profound deafness of a perception type. It is said that bizarre findings are seen, such as loss of reaction to the caloric test but retention of reaction for rotation and vice versa. The significance of these findings is disputed by others. In Vienna, for a long time a shortened bone conduction in the presence of fairly good hearing was considered to be pathognomonic of syphilis, and this diagnosis was made at times with no other findings present or even sought.

Treatment of syphilitic deafness of the perception type, either congenital or acquired, is disappointing with the exception, according to Mackenzie, of the type seen in cerebrospinal syphilis with meningitic involvement. Vigorous therapeusis will often give favorable results.

It would seem that with the careful prenatal care given in large parts of the country, the infantile, or prenatal, and the late hereditary types will gradually disappear. As for the acquired types of syphilitic deafness, the present campaign against the disease in general in this country will, no doubt, cause us to see fewer numbers of this type as time goes on.

Laryngeal Lesions

JOHN DELPH, M.D.

(Abstract)

Syphilis as seen in the larynx is a local manifestation of a general disease. No accurate statistics are available on the incidence of syphilitic laryngitis, although with its relation to diseases of the larynx in general, it is believed to occur in about 6 per cent of cases, and to be more prevalent in males than in females.

The primary lesion is rarely encountered in the larynx, although there are reports in the literature of chancre of the epiglottis. This is explained by the fact that the initial lesion is almost always single, and inoculation, by kissing for instance, is almost sure to attack the mucosa higher up in the respiratory tract. Also, the laryngeal mucosa is well protected from any invader except those that are air-borne.

The lesions of the larynx were described in the various stages. The chief symptom in the early stages is hoarseness. There is usually no pain or cough. Diagnosis cannot be made on the basis of symptomatology or previous history only. It has been said that if one could observe enough cases, he would learn to suspect syphilis from the characteristic appearance of the mucosa. The primary considerations in differential diagnosis are tuberculosis and malignancy. Examination of a biopsy specimen should be made if there is any question.

Treatment of syphilis of the larynx, hereditary or acquired, should be left to the syphilologist, and the laryngologist should confine himself to observation and be prepared to meet any emergency that may arise. The prognosis is usually satisfactory unless the cartilage has been involved, in which case surgery may be necessary. Unless the ulcers are in the subglottic space or in the region of the arytenoid articulation, there is little danger to life.

Dermatological Reactions in the Treatment of Syphilis

JOSEPH WELFELD, M.D.

(Abstract)

The cutaneous aspects of syphilis and its reactions, and common skin affections simulating syphilis were described as encountered at the Municipal Social Hygiene Clinic where approximately 1500 cases of syphilis are seen daily. Any patients exhibiting the skin manifestations are referred to the dermatological department for a complete check-up before any treatment is instituted. Those who have received treatment and show reactions are sent back to that department where their future therapy is determined.

The mechanism of drug reactions is not known. The drugs may act directly on the skin or may act indirectly by primary effect on the liver. It is known, however, that in most instances a definite hypersensitiveness of the skin may exist.

Drug eruptions are sometimes difficult to distinguish from other cutaneous manifestations. The diagnosis can only be established by careful questioning, history of taking drugs, improvement of skin lesions when administration is stopped and by testing the patient's sensitiveness to the drug.

The eruptions fall into four general categories: vascular reactions, eczematous reactions, follicular reactions, and ulcerating and vegetating eruptions.

It is found that these patients exhibit typical lesions of neurodermatitis, and it has been concluded that cases of pruritus with lichenification must have some relationship to syphilis because so large a per cent improve when placed under anti-syphilitic therapy.

The classification is composed of two groups: cutaneous and constitutional reactions. Reactions should be anticipated when a patient presents a history of previous dermatoses such as pruritus, urticaria, eczema, hay fever, and asthma.

Reaction frequently occurs after the first injection, and at times there seems to be a hypersusceptibility created by long rest between injections. Mild eruption will appear such as erythema, morbilliform, scarlatiniform, or eczema, followed by desquamation. This is usually of short duration and undoubtedly is due to anaphylaxis.

The constitutional reactions are divided into three groups: immediate, slightly delayed and delayed.

Immediate nitroid reactions occur during the injections or immediately afterward. There is a sudden vasomotor disturbance in the train of symptoms.

Slightly delayed reactions are due to arsphenamine, and are symptomatically described as follows: nausea, vomiting, diarrhea, chills, headache, urticaria, fever, and joint pain. They require no treatment unless they are prolonged and continuous.

In delayed reactions there are numerous types of dermatitis, but the principal object is to avoid the vesicular and exfoliative cases which cause long periods of invalidism and may result in death.

DISCUSSION

DR. JAMES H. MITCHELL: I have been in close touch with Dr. Senear's work for a long time, and have had the pleasure of reading his paper which appeared in the collected papers of the symposium of the Association for the Advancement of Science at the Indianapolis session. I think all who are interested in the subject would do well to read this paper. In 1926 I read a paper at the Dallas meeting of the American Medical Association showing what terrible things can result from false positive reactions; there were some rather tragic

episodes in that report. I think we should all bear in mind the possible tragedies that may occur as a result of false positives.

Dr. John E. Muslow: About 18 years ago, before the Wassermann test was compulsory at my clinic, I became interested in the matter of syphilis with regard to chronic infection of the ear, nose and throat. Wassermann tests done at that time on 370 unselected patients revealed a 4-plus in 65, a 2 and 1-plus in 24 patients. Among the group having a 4-plus Wassermann were a fairly large number of patients who had had several operations for various chronic sinus conditions with no definite improvement. Included in this latter group were several chronic ear and laryngeal conditions which had not responded to conservative treatment. There is one case that comes to my mind particularly. A man had been referred to the ear clinic with a diagnosis of a brain abscess. Examination showed a profuse greenish, malodorous discharge from the left ear. The Wassermann test was 4-plus. Under anti-syphilitic treatment, combined with conservative treatment to the ear, the condition healed. He had normal hearing in this ear when discharged. We are all familiar with the mucous patch in the mouth; why should not one also think of a mucous patch in the antrum, eustachian tube or in the middle ear? A likely complication would be a superimposed pyogenic infection and perhaps bone destruction. I feel it is imperative to obtain a Wassermann test in all chronic infections of the ear, nose and throat that do not respond to the ordinary methods of treatment. As Doctors Lawson and Mitchell have stated, the atrocious tertiary lesions of the nose and throat are seldom seen today. This is especially true in the past ten years of my experience.

Dr. Sherman Shapiro: I should like to add a few words to Dr. Pearlman's very able presentation of syphilis of the ear, particularly in reference to that of the inner ear. With regard to shortened bone conduction, I think most of us are now agreed that this finding is simply evidence of profound inner ear involvement, and may be found not only in syphilis but in many other types of inner ear deafness. I was interested in what he said about the so-called paradoxical labyrinthine reactions. I have seen this in syphilis as well as in other conditions. It is supposed to be due to a retro-labyrinthine process. The symptom is of great interest, and I do not know of any adequate explanation for it. An occasional finding which was not mentioned is the Hennebert fistula sign reaction which is practically pathognomonic for syphilis of the inner ear.

Dr. Walter Theobald: Referring to the case Dr. Mitchell showed, in which a tongue was treated for malignancy which later

proved to be syphilis in the absence of a positive Wassermann, I should like to ask a question. What, if anything, can Dr. Mitchell tell us about provocative treatment or provocative manifestations? That is, when one is in doubt, is there any means of provoking the blood so we can find a positive Wassermann?

Dr. Howard C. Ballenger: In discussing the complications of the treatment of syphilis by means of the arsenicals and mercury, the sudden onset of deafness which was formerly seen was not mentioned. Other cranial nerves have been reported as being involved at times as well. When these cases were mentioned several years ago, it was thought to be due to faulty preparation of the drug or to use of insufficiently large doses. This complication does not seem to be so common in recent years. I wonder if any of the speakers have observed this result from the use of the arsenicals.

Dr. James H. Mitchell: I think Dr. Senear will agree that the provocative reaction is of great value in dermatology. It has been known for a long time that from 25 to 30 per cent of late cases give a negative reaction. The procedure consists of giving a small dose of neoarsphenamine or old arsphenamine, and then waiting for three weeks. Leredde, who first proposed this, suggested a three weeks' period in view of the fact that the Wassermann is quite slow in becoming positive in primary infections. Some advise testing the blood the next day, or within three days, but in my experience it is better to wait three weeks. In that way we allow plenty of time to elapse for the Wassermann to become positive, and also allow ample time for a therapeutic result. We get both a therapeutic test and a serologic test, and it has been of great value in my experience.

Dr. Joseph Welfeld: Fixed pigmentations have entirely different characteristics than those mentioned by Dr. Mitchell, which are caused by practically the same drugs. We have grouped those cases separately, and they have been observed by other dermatologists as a true arsenical melanosis.

We can say that a large per cent of cases develop all characteristics of neurodermatitis, which I emphasized. Are they due to syphilis? I presume there must be some relationship to syphilis because they improve under arsenical therapy.

Dr. F. E. Senear: Dr. Welfeld spoke of the occurrence of hyperkeratotic lesions. I do not see them occurring as a result of syphilis in my experience. We do know that lichenification is seen

in colored patients to an extent that it is not seen in white. Arsenic is an old remedy in the treatment of hyperkeratosis, and I would feel it might have an effect on dermatitis, but would feel it was nothing but the indirect effect of therapy.

In reply to Dr. Welfeld's question, I have not seen any type of simple lichenification of neurodermatitic type which I could attribute to syphilis. I did not understand whether or not you attributed that to syphilis. My answer is that I have never seen it.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, March 4, 1940

THE PRESIDENT, DR. GEORGE T. JORDAN, IN THE CHAIR

Pituitary Dwarfism With Atrophic Rhinitis*

NOAH FABRICANT, M.D.

AND

PAT BRONSTEIN, M.D.

(Abstract)

Children exhibiting sexual infantilism and defective growth (Lorain-Levi dwarfs) may be normal at birth and develop adequately until some time in childhood, when growth either ceases or becomes retarded. No special organs are involved. The proportions are those of a normal child. The genital retardation is probably not in excess of the physical development, and the secondary sexual characteristics are markedly delayed or absent. The mentality may be normal, although the behavior is often immature.

No gross evidence of dystrophy of the bones or joints exists, although chondro-epiphyseal changes, poorly outlined osseous centers with decreased bone density, have been described. The ossification centers usually appear at the normal time, whereas delay in epiphyseal closure is a consistently positive finding. The basal metabolic rate may be normal, but some question exists where there is a co-existent mild hypothyroidism which may induce slight delay in carpal development and moderate reduction of the basal metabolic rate.

A pituitary dwarf aged 15 with sexual infantilism (Lorain-Levi), exhibiting two unusual findings, a calcified lesion in the sella turcica and an atrophic rhinitis, is described. Rarefaction of the tibia is noted.

---

*From the Departments of Pediatrics and Otolaryngology, University of Illinois College of Medicine and the Research and Educational Hospitals.

Estrogens which have been employed by others in atrophic rhinitis were not used in our patient because of the possibility of premature epiphyseal closure.

The irregular administration of growth substances over a period of approximately two and a half years presumably produced a gain in weight and an increase in height, but apparently had no effect upon the atrophic rhinitis.

DISCUSSION

DR. I. PAT BRONSTEIN: It is unusual to be able to show a Lorain-Levi dwarf in whom the diagnosis is more than presumptive. This patient presented a calcified mass in the sella turcica and, in addition, atrophic rhinitis. The case is important since it raises the question of the use of various hormonal products. I have had occasion to go over your literature in cursory fashion and have noted the increasing number of articles on the endocrinologic aspects of ear, nose and throat diseases. I should like to stress some basic facts. This child is a dwarf with two conditions—undersize and sexual underdevelopment. A number of authors have advised estrogenic material for atrophic rhinitis, and if one uses this unwittingly in this type of child, it may defeat the more desired purpose of increasing growth. The possibility of premature closure of the epiphyses by the excess use of sex hormones is of sufficient importance to warrant caution in their use.

I have had in the last two months two experiences in preadolescent children where an excess of sex hormones was found. One, a child aged about 11, a pseudo-hermaphrodite, had an excess of androgenic substances in the urine with the epiphyseal closure practically completed. Nature can thus produce what we accomplish artificially by giving excess amounts of these products.

Slides were shown of two other conditions of otolaryngologic significance:

1. A child with a huge tongue was referred as a cretin because of the macroglossia. Biopsy revealed a true muscular hypertrophy.

2. Two cretins were shown because of deafness. One was untreated until 10 years of age, and possibly the deafness represented an end stage of cretinous degeneration. The other was a child treated actively and adequately since the age of six months. Here the etiology of the deficient hearing is not so clear.

Carcinoma of the Esophagus in Association With That of the Tongue

J. SCHOOLMAN, M.D.

(Abstract)

The first case shown had had a resection of the right half of the tongue because of malignancy twenty-two years ago. The patient recently came into the clinic complaining of difficulty in swallowing. Examination of the floor of the mouth revealed marked scarring, but no recurrence of the lingual lesion. Esophagoscopy revealed an obstructive tumor mass involving the upper one-third of the esophagus half way between the cricoid and the aortic notch. On biopsy this proved to be an anaplastic squamous cell carcinoma. Gastrostomy was advised, but the patient left the hospital without permitting this procedure.

The second case was sent into the hospital for tracheotomy because of acute respiratory distress. Examination revealed a tumor mass at the base of the tongue and an infiltrating mass obstructing the trachea above the bifurcation. Questioning brought out the fact that the lingual tumor had been treated. Autopsy revealed a hornifying squamous cell carcinoma of the tongue, containing a radon seed. The second tumor was an ulcerated squamous cell carcinoma of the esophagus, which perforated into the trachea and metastasized to a perigastric lymph gland.

Two questions were discussed: Is the tumor of the esophagus secondary to that of the tongue, and, if secondary, was the process that of metastases or implantation? Are both the tumors primary?

In both the cases cited the lungs were free. An extension from the tongue to the esophagus must pass through the deep cervical glands and continue against the lymph current draining the esophagus. In our second case both lesions were present at the same time. The time factor in our first case also serves to discount the question of metastasis, the period between tumors being more than double that which Holden reports as having elapsed between the removal of a breast carcinoma and the appearance of bone metastases.

Until it is definitely settled whether implantation can occur in the esophagus, we may assume that we are probably dealing with multiple primary tumors, and conclude that multiple primary tumors do occur in a related organ-system, and the cure of one lesion should keep one alert for the possible appearance of another even after a prolonged interval.

Neoplasms of the External Auditory Canal

M. F. SNITMAN, M.D.

(Abstract)

The cases demonstrated were seen rather late in the course of the disease and, as a result, it is difficult to determine definitely whether they are of middle ear or external canal origin. However, from a study of the history and the histopathology it would appear that the majority are of external auditory canal origin.

From 1925 until the middle of 1939 eight cases of carcinoma of the external and middle ear were observed. During the latter months of 1939 five cases of carcinoma of the external auditory canal were seen. On two occasions, four of the five were hospital patients at the time. All the patients were women, the youngest 25 and the oldest 51 years of age. In four, the lesion was in the right ear, while in one case the left ear was involved. All were primary lesions.

The histopathology of each case was presented.

The essayist emphasized that improvement in these cases can be obtained early if due attention is given to any growth or polyp in the canal or middle ear. Early biopsy should be the rule here as elsewhere. It is distinctly advantageous for the clinician to have an understanding of pathology, since it enables him to co-ordinate clinical knowledge with the biopsy specimen. For the pathologist this is usually not feasible.

Fatal Hemorrhage After Puncture of the Maxillary Sinus

JOHN WALL, M.D.

(Abstract)

A man, aged 54, stated that about three weeks prior to examination he noticed a soreness over the right side of his nose; there were no symptoms. The following day, severe pains developed over this area and spread to the right side of the forehead, the right eyeball, and over the right parietal region. He had a feeling of weakness accompanied by generalized muscular pain, more pronounced

in the back. Several days later, he developed a puffy right eye and began to see double upon looking forward and to the left.

When first seen, the right eye showed an external third nerve paralysis with a moderate exophthalmos; the pupils reacted to light and accommodation; the conjunctiva was sensitive. The nose was apparently normal, except for a moderate deviation of the septum. The epipharynx contained only a small amount of mucoid discharge. Transillumination of the sinuses revealed a clouding of the right frontal and maxillary sinuses. X-ray corroborated the transillumination.

A puncture of the right antrum was made under the inferior turbinate, and a small amount of pus with much bleeding was obtained through irrigation. In making the puncture a soft lateral nasal wall was felt, which gave the impression of a soft tumor-like mass within the antrum. The extreme pallor of the patient and his back pains in addition to these findings pointed to a carcinoma with multiple metastases in the skeleton, or a myeloma. That afternoon, severe epistaxis occurred, but this was easily stopped with nasal packings.

The patient continued to show signs of internal bleeding, his pulse became thready and rapid, and his blood pressure dropped. He then began to vomit large amounts of fresh blood. Repeated examination of his nose failed to show any bleeding, and a hemorrhage originating in the stomach was considered. The symptoms continued, and in spite of two blood transfusions and intravenous fluids, he gradually became weaker, comatose and died the following morning, about 24 hours after the puncture of his sinus.

Autopsy showed a large carcinoma of the stomach and generalized carcinomatosis of the dura. These multiple meningeal metastases were particularly present in the region of the superior orbital fissure, and were the cause of a large depression in the temporal lobe of the brain. There was no gross tumor tissue in any of the sinuses.

Additional sections made of the original stomach tumor and of the meningeal metastases showed them to be identical with the tumor tissue found in the sinuses. Active mitosis could be seen in some of the cells.

An effort was made to correlate the severe gastric hemorrhage and rapid death of the patient with the antrum puncture. It is stated in the literature that death may follow antrum puncture either from an air embolus, cocaine poisoning, or a reflex irritation of the vagus nerve, which may be stimulated by branches of the first or

second division of the trigeminal nerve. It has been proved that there is a definite connection with the fifth nerve and the vagus, and that the latter can be stimulated or irritated at will with irritants in the nasal or antral mucosa. It was assumed that since the blood vessels of the stomach already had been damaged by the carcinomatosis the vagal irritation, by causing some disturbance in the circulation, started the active hemorrhage. The vagus nerve in turn was stimulated by the nasal reflex through the branches of the fifth nerve in the antrum mucosa. This was a direct result, however, of the antrum puncture and irrigation.

DISCUSSION

DR. HANS BRUNNER: The two cases discussed by Dr. Schoolman lead to the difficult and important question as to whether one is concerned with two primary tumors or with one tumor and implantation metastasis in the esophagus. In his very logical comment Dr. Schoolman decided in favor of the possibility of multiple primary carcinomas. Nevertheless, I should like to emphasize one point which can be used in behalf of the diagnosis of implantation tumors in the esophagus. Many decades ago it was found that the mucous membrane of the esophagus contains glands of a gastric type, namely, glands which probably produce pepsin. These glands are found only in the cardiac portion of the esophagus and in its uppermost portion, between the cricoid cartilage and about the fifth tracheal ring. It is obvious, then, that in these areas implantation carcinomas can but rarely develop because of the digestive power of the gastric juices. But there is no inhibition to the development of primary carcinomas in these areas. It is noteworthy that in both cases reported by Dr. Schoolman, the tumors within the esophagus avoided these areas, a point which is suggestive for the diagnosis of implantation tumors.

The case reported by Dr. Wall is of great importance because it acquaints us with a disease of the mucous membrane of the nose which has been suspected but heretofore not proved. In this disease we find a great part of the capillaries and the lymph vessels of the mucous membrane filled with emboli of carcinoma cells. This finding is well known in the pleura and lungs and in the dura, but not in the nasal mucous membrane. According to these findings, we must call the disease described by Dr. Wall "carcinomatosis of the nasal mucous membrane." Clinically, we are as yet not able to make a diagnosis of that disease. However, since it may be assumed that this disease is not so extremely rare, it is our next duty to discover the clinical symptoms of carcinomatosis of the nasal mucous membrane and establish thereby a clinical as well as a pathologic entity.

DR. JOSEPH C. BECK: I was astonished to hear one of the essayists, Dr. Snitman, say that in histologic examinations we should not take heed of the pathologist's viewpoint. I think that is an erroneous assumption. Granted that the pathologist does not know the clinical picture, it has been my plea for many years that clinicians should study pathology; but still we need the pathologist. I should like Dr. Wall, when he mentions carcinomatosis, to give us the identity of that cell that he demonstrated. What is the type of cell that he diagnosed as carcinoma? One variety is anaplastic, another is not so. However, the pathologist is essential in making the diagnosis with us. We can give him the clear clinical picture, and he is of greatest aid.

To me these three cases that have been presented and all the work that has been done have been a treat; to see the ease with which these pathologic diagnoses were made by these youngsters is wonderful. We are going ahead by leaps and bounds, and pretty soon there will be no failures by the Board (maybe)—certainly not in pathology. I want to compliment the department on this excellent presentation.

DR. J. SCHOOLMAN (closing): I would like to thank Dr. Brunner for his discussion and for allowing me to take advantage of his comments in the department, and the thought I have been able to put on it outside the department. In referring particularly to this gastric type of gland which may lodge in the esophagus, it will be remembered that the detached tumor cell has to pass an area of gastric digestion, and will be less likely to implant disease into even the weakened area of resistance in the esophagus below it.

DR. M. F. SNITMAN: I agree most heartily with what Dr. Beck has said, but I did not mean to imply that we should not work in conjunction with the general pathologist. However, it is to the advantage of the clinician to interest himself in the study of the disease tissue, because he is thus able to coordinate the clinical picture with the histopathology. This has been well borne out in the experience of Dr. Brunner and myself.

DR. J. WALL (closing): This tumor was recognized and diagnosed as carcinoma simply upon the highly differentiated cells, and as long as there was no outstanding feature we diagnosed it as we would other carcinoma cells. This condition may be unrecognized as this one would have been had not Dr. Brunner asked us to go further with it. It was accepted as involving the meninges alone, and only on Dr. Brunner's insistence was it further studied.

Intravenous Feeding With Special Reference to Amino Acids

ALBERT H. ANDREWS, JR., M.D.

(Abstract)

The usual intravenous administration of water, salt and carbohydrate is inadequate for prolonged therapy because of the absence of protein. Intravenous administration of a 5 per cent solution of the essential amino acids* is a proposed method of maintaining an adequate protein intake. Transient reactions consisting of fever and chills occur if the administration is too fast, but no harmful effects have been observed after the reactions subsided.

Sufficient data on the clinical use of essential amino acids solutions has not as yet accumulated, but the evidence so far indicates that it does supply protein in a utilizable form. Its clinical use is indicated at the present time in those cases in which prolonged intravenous feeding is essential for the maintenance of life.

DISCUSSION

DR. PAUL HOLINGER: The problem of feeding a patient dehydrated because of esophageal obstruction has been a desperate one as long as there has been esophageal obstruction. This recent advance that Dr. Andrews has presented has been a major advance in therapy. The first step usually taken is that of gastrostomy. However the percentage of mortality from gastrostomy in dehydrated and cachectic patients is extremely high, and it is only by resorting to other means than immediate gastrostomy that the percentage can be lowered. Patients generally have a protein lack not only because they have been completely obstructed for a few days prior to hospitalization but because they have not been able to take adequate quantities of proteins for weeks or months previously. Increasing the fluid and carbohydrate intake alone has certainly been augmented by the addition of intravenous amino acids. The improvement and sense of well-being the patient displays are gratifying to observe and much more pronounced than the well recognized reaction to saline and glucose alone.

---

*Supplied by the Baxter Laboratories, Inc., Glenview, Illinois.

Osteomyelitis of the Frontal Bone: Sine Frontal Sinusitis

ROBERT B. LEWY, M.D.

(Abstract)

It is well recognized that osteomyelitis of the frontal bone may occur, complicating small pox, as a manifestation of a hematogenous extension of a generalized osteomyelitis affection, or secondary to trauma, but its occurrence in the absence of a predisposing frontal sinusitis must be quite rare. This probably explains its infrequency prior to pubescence and the time of life of the well developed frontal sinus.

Two cases are herewith presented in detail, in one of which (a woman aged 44) the outcome was fatal. The other (a girl aged 8) is still under observation. The localization in the outer table in the young patient herein described is most probably explained by the greater porosity of the outer table of the bone as opposed to the more vitreous character of the inner table. This case represents a special therapeutic problem since it does not require the usual sinus approach, and it is in close proximity to the superior sagittal sinus. It is of interest in the same case to note that the process developed in the midline at a point away from the place of origin of the frontal sinus.

The other case doubtless represents (in retrospect) the fulminating type of Hagelman and Williams. Here no surgery could have been adequate to stop the advancing lesions of the infection.

DISCUSSION

DR. SHERMAN SHAPIRO: These very interesting case reports speak for themselves. One may say they illustrate as no didactic discussion can the very important role of the diploetic layers of the cranial bones in the production of osteomyelitis. A few words as to classification: One gets the impression that a good many men consider only a spreading osteomyelitis as a true osteomyelitis of the cranial vault. I prefer the classification given by the essayist, not only because it is in accord with clinical experience but also because it is in line with the histopathology of this condition. Particularly in recent communications it has been shown that along with the destructive and necrotic processes that take place in osteomyelitis of the cranial bones, there is also a reparative process resulting in granulation tissue and bone, and sometimes this process proceeds to

actual osteosclerosis. One gets the impression that the prognosis depends pretty much on this reparative process.

I recall a particularly interesting illustration of this fact. About two or three years ago we had a nurse sent into Research Hospital from downstate, with a history of chronic infection of the occipital bone for three or four years, arising from a mastoid infection. She had a mastoid fistula dating from a previous operation, as well as an occipital fistula near the midline. It was decided to connect the two fistulae by removing the occipital bone between them. At operation not only was the occipital bone found to be markedly thickened—three-fourths to one inch—but the diploe was obliterated—this being an extreme example of the reparative process. This woman, by the way, made a complete recovery.

A practical point we can draw from this classification is that in those cases where the clinical and x-ray findings indicate a localizing process, there is no need for the very extensive procedures that are done in spreading osteomyelitis, and the patient will probably get well with more conservative measures.

The causative organisms in the case just cited were streptococcus hemolyticus and staphylococcus aureus. In another case of cranial osteomyelitis following a simple mastoidectomy—one that ended fatally—a streptococcus mucosus was responsible.

DR. T. C. GALLOWAY: One factor that is often overlooked is the nature of the infective organisms in the production of osteomyelitis. Mosher, Furstenberg and others have definitely worked out in the diploic bone the role of the staphylococcus, which produces extension by invading the veins. The disease can extend through apparently sound bone to break out under the periosteum or over the dura at some distance. The only safe treatment is radical removal.

The processes that begin around the antrum seem different. I have seen four of these in the last year, three of which were fatal. Why should a process which begins in relation to the cancellous bone of the antrum be so hard to arrest? There are no easily infected vascular channels. In three of the cases cited, the infecting organism was an anaerobic slightly hemolyzing streptococcus, associated with much soft tissue swelling and adenopathy which might suggest a cellulitis with lymphangitis. It is hard to get good anaerobic cultures, and routine cultures unless made in the depth of brain broth will not pick them up. My last case developed following a simple

antrum window. It looked as if it would be fulminating and rapidly advanced as long as I waited. It was, however, quickly arrested by the technic advised by Meleney for anaerobic streptococci. An oblique incision about 2 cm. was made down and out from the lacrimal fossa, the periosteum was freed under the whole infected area, a 0.5 cm. button removed (with a laboratory report of osteitis) and the soft tissues freed intranasally over the inferior turbinate. Twice daily for one week, freshly prepared 30 per cent paste of zinc peroxide in water was injected into the undermined areas. No further extension occurred, and healing was relatively rapid. Sulfapyridine had been given but was stopped on account of a rash before this intervention.

DR. ROBERT LEWY (closing): I am very appreciative of the discussions of Dr. Galloway and Dr. Shapiro. I think the question of bacteriology of osteomyelitis of the frontal bone is subject to considerably more scrutiny than it has had in the past. In the one case where the infection was in the maxillary sinus and wound up in the frontal bone, it is hard to trace the pathway by which such infection can travel. This case developed a septal abscess and from that point osteomyelitis of the frontal bone. Perhaps the ethmoid was involved. We never found any particular involvement, but if there were, it might support the theory that the frontal is nothing more than an enlarged ethmoid cell, and therefore the course of this infection is much more logical than it would seem.

Retropharyngeal Fibroma and Tertiary Lues: Case Report*

L. Z. FISHMAN, M.D., M.S.

(Abstract)

This 35-year-old white man was first seen in December, 1936, with a complaint of cough, difficulty in swallowing and breathing, swelling of the neck, and a loss of thirteen pounds in weight in a period of seven months. A tentative diagnosis of carcinoma of the esophagus was made at the time of admission.

Because of the positive serologic reports, anti-syphilitic therapy was instituted and continued for a period of more than two years.

*From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, and Research and Educational Hospitals, Chicago.

In February, 1939, a mass weighing 345 grams was removed from the retropharyngeal space.

Indirect laryngoscopy on the tenth postoperative day revealed both cords fixed: the left markedly abducted and the right in the "medio" paramedian position. The right vocal cord evidenced slight motility at the time of discharge on the 27th postoperative day; the left remained in the "medio" paramedian position.

A correct diagnosis had been made one year prior to the removal of the tumor by means of an exploratory operation on the neck for the purposes of obtaining a biopsy specimen. Despite this, surgical treatment was delayed because of the coincidental tertiary syphilis.

Although there was considerable prolonged pressure against the posterior surface of the larynx and trachea to the extent that the latter was compressed to a narrow slit, there was no resulting chondromalacia. There was no difficulty in entering the narrow lumen of the trachea and passing a cannula beyond the seemingly impassable obstruction. The lumen of the trachea was completely restored following the extirpation of the tumor.

Microscopic studies of a number of sections taken from different parts of the mass and by selective methods of staining proved the tumor to be a benign fibroma durum; one position is suggestive of a leiomyoma or fibroma molle. In either event, any attempts to correlate the pathogenesis of this fibroma with the patient's luetic infection can only be a matter of conjecture.

Of interest is the unusual size of a fibroma of the retropharynx and the extensive exposure occasioned by its removal in such a hazardous region. The reparative response of the tissues of this patient was not disturbed by his previous luetic infection.

Syndrome of Petrous Tip

W. Loeb, M.D.

(Abstract)

This patient is a white woman, aged 30, whose present illness began in September 1939. She contracted a respiratory infection, accompanied by pain in the right ear. There was elevation of temperature, a nonproductive cough, and diffuse headache. Paracen-

tesis of the right ear was performed four days after the onset, following which the temperature returned to normal, where it remained during the ensuing course of the illness. A second paracentesis was performed three days later. There was moderate purulent discharge from the right ear, but the pains persisted.

Three weeks after the onset, the ear was still draining; there was right-sided fronto-parietal headache, and some pain deep in the right eye. The patient then noticed double vision on looking to the right. The pain in the right eye increased, and the diplopia persisted. In the sixth week of the illness she complained of gradually increasing rotational dizziness, occurring intermittently.

Examination showed a draining central perforation of the right drum, sixth nerve paralysis on the right, and second degree nystagmus to the left. There was no sagging of the canal wall, no mastoid tenderness, and no tip edema. The hearing in the right ear was 12 feet whispered voice; the Weber lateralized to the right; the fistula test was negative. The left ear was normal. General physical examination was negative. The blood Wassermann was 4+, the patient having had two years' continuous anti-syphilitic therapy. Spinal tap revealed clear fluid under normal pressure, three white cells, and no increased protein. The serology was negative.

X-rays showed a clouding of the mastoid process, and no petrous tip could be made out on the right side. A diagnosis of subacute mastoiditis and petrositis was made. An attico-antrotomy was done, revealing granulations in the antrum, over the horizontal semicircular canal, and eroding the tegmen. The dura was exposed, and was covered with granulations. The lateral sinus was also exposed and appeared normal.

One month after operation, the patient felt well and had gained weight. The abducens paralysis was slowly diminishing. There was continued discharge from the right ear, but no pain. Spinal tap was again done to check the serology. At this time there were 30 leucocytes, increased protein, and negative serology. Shortly after she complained of occasional severe pain deep in the right eye. The question then arose as to whether the continued abducens paralysis and headaches were due predominantly to syphilitic meningitis, or to petrositis. Further x-rays revealed a continued rarifaction of the petrous tip on the right side.

Nine weeks after the first operation, the patient was readmitted to the hospital. Examination showed diplopia on looking to the

right only, no nystagmus, 20 feet whispered voice in the affected ear, Weber lateralized to the affected side, and negative neurologic examination. Spinal fluid showed three cells, no increased protein, and negative serology.

The petrous apex was approached by the epidural route, removing the roof of the attic and eustachian tube, and reflecting away the dura. No pus was found, but soft bone suggestive of a healing osteomyelitis. This was corroborated by the microscopic sections. The incus, lying loosely in granulation tissue, was removed. One hour postoperatively, the patient was seen to have a complete facial paralysis on the right. This is slowly subsiding one month after operation. The abducens paralysis was completely gone by the third postoperative day. The course was otherwise normal.

Six weeks after the second operation, the patient has two-foot whispered voice in the right ear, with the Weber lateralized to the right. There is no sixth nerve paralysis.

The case illustrates the difficulty of determining whether the cranial nerve involvement was due to syphilis or petrositis and is an example of the difficulties encountered in the diagnosis of petrous abscess by x-ray.

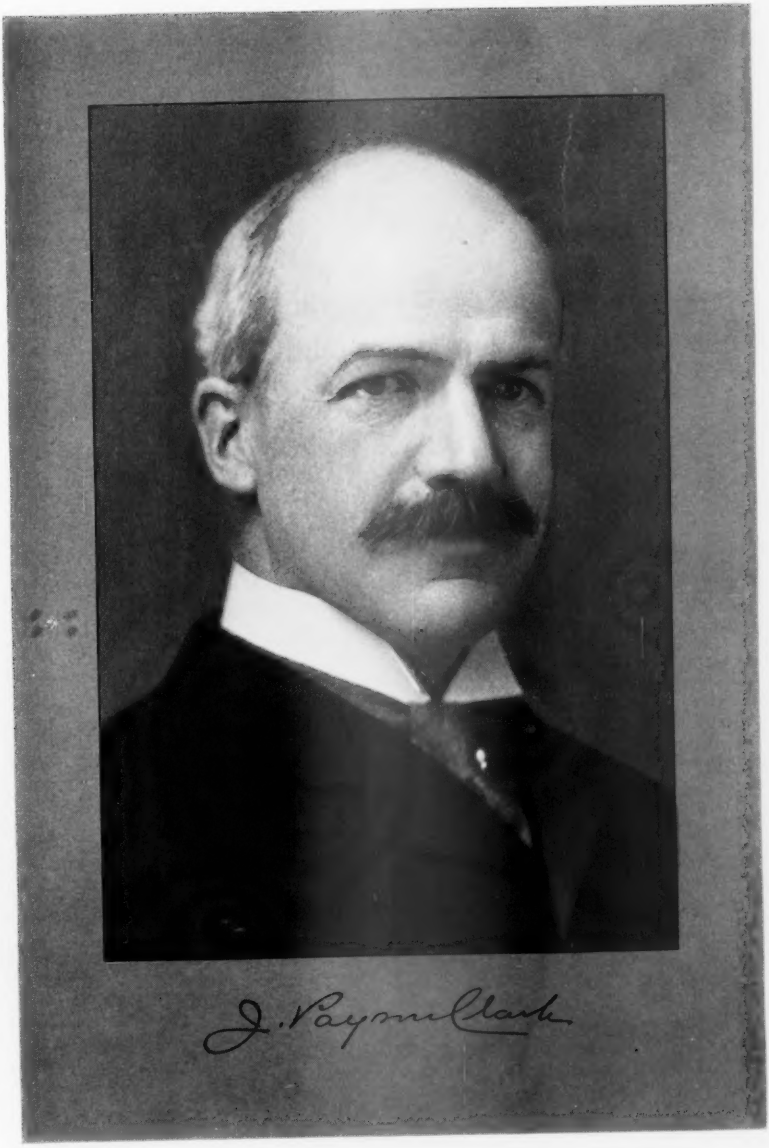
DISCUSSION

Dr. T. J. Wachowski: This case presented two problems, the first relating to the differential diagnosis of the very obvious pathology seen on the films. In this respect, the x-ray department was not able to aid much because either a gumma or an advanced petrositis could produce the destruction seen.

The other problem related to the diagnosis of petrositis per se. This was more difficult because of, first, the position of the petrous bone in the center of the skull, where it is inaccessible for sharp radiography; and, second, the fact that, in the adult, the petrous area is built much like the mastoid areas in the child, that is, cellular air spaces within a medullary framework. The changes that occur may be due to asymmetry of development, some of which is probably related to middle ear infections in childhood. In inflammatory pathology, the changes differ in the acute and chronic stages. In the acute stage, there is increased density due to loss of air spaces secondary to an empyema of the cells. These early changes are difficult to determine unless one has an earlier "Scout" film for comparison, and I believe that it is advisable to make such a film, preferably in the

Taylor position, in all cases of mastoiditis. As the pathology progresses, there is loss of calcium in the area due to a type of osteomyelitis which develops. This gives rise to areas of decreased density. While these areas may occur anywhere, they are more common in the very thin apex of the petrous bone, and for that reason, the Stenvers position is most suited for showing them. Other views, such as the Mayer position, to show cells at the postero-medial angle, may be necessary for a full appreciation of the pathology. As a base film for future reference, however, I believe the Taylor position is adequate.

Dr. Hans Brunner: Insofar as prognosis is concerned, there is a great distinction between the different kinds of petrositis. It is my experience that the most dangerous of these types are those in which the symptoms of the petrous apex become evident in the late stage of an acute otitis media, viz., in the fourth to the sixth week. This very thing occurred in the case discussed by Dr. Loeb. Furthermore, since the x-ray examination revealed a definite destruction of the petrous apex, the operation was considered imperative. The normal appearance of the drum and good hearing at that time indicated the subdural approach and contraindicated the operation after the technic of Ramadier. The operation was difficult, inasmuch as there was no tract leading to the apex. No granulations and no pus could be found. However, the bone was removed from the roof of the middle ear and eustachian tube and from the anterior surface of the pyramid up to the gasserian ganglion. During this procedure the geniculate ganglion was injured, resulting in a postoperative facial paralysis. The removed bone did not give the impression of healthy bone, and the microscopic examination of it revealed an osteomyelitis and osteitis, healing with the production of osteoid bone and connective tissue. As neither osteoid bone nor connective tissue produce a shadow in an x-ray study, the roentgen film evidence in this case is therefore readily explained. After the removal of the bone, the headache and the abducens paralysis disappeared. It is probable, however, that they would have disappeared without operative intervention. Consequently, we draw the conclusion that all symptoms of the petrous apex, including a positive finding on x-ray examination, can be the result of a healing osteomyelitis and osteitis of the petrous apex. Considering the great tendency of all types of petrositis to spontaneous healing, this case again proves that operations upon the petrous tip are seldom necessary, a point of view which I have held since 1926, when I became especially interested in such cases.



J. Paynter Clark

JOSEPH PAYSON CLARK

1860-1940

Joseph Payson Clark, prominent in laryngological circles at the beginning of the present century and at one time president of the American Laryngological Association, died at his home in Boston, Massachusetts.

He was the son of Joseph J. Clark and Mary Elizabeth Mulliken Clark of Richmond, Virginia. He was born in St. Louis, Missouri, on April 16, 1860, where he spent his early life, but since his maturity he lived at Roxbury, Massachusetts and Boston.

He was graduated from the Harvard Medical School in 1887 and after a surgical interneship in the Massachusetts General Hospital, began the study of laryngology at Vienna under Chiari and Schnitzler. Upon his return he became associated with the Massachusetts General Hospital, serving as Assistant Laryngologist there from 1889 to 1893, and as Laryngologist from that time until 1916 when he resigned that position. During the First World War he returned to service there for two years, relieving those who had joined the armed forces.

Doctor Clark contributed abundantly to the literature of his time. His reports were entirely of a clinical nature. They appeared over a span of years from 1890 to 1911 and dealt largely with diseases of the pharynx and larynx.

AUSTIN ALBERT HAYDEN

(1881-1940)

Austin Albert Hayden was born in Shullsburg, Wisconsin, October 15, 1881. He died in Chicago, July 10, 1940.

After the usual secondary education he attended Creighton University, Omaha, the University of Chicago, and graduated from Rush Medical College in 1904.

Leading a very active life until his death from coronary thrombosis, he had many and varied interests. He was an instructor in Ophthalmology at the New York Post-Graduate Medical School 1906-1908, and at Rush 1908-1916. He was a Fellow of the American College of Surgeons, a member of the American Otological Society, the American Academy of Ophthalmology and Oto-Laryngology, the American Laryngological, Rhinological and Otological Society, the Institute of Medicine, and the Society of Medical History of Chicago. He was a former President of the Chicago Laryngological and Otological Society, the Chicago Medical Society, and the American Association of Railway Surgeons.

He was President of the Chicago League for the Hard of Hearing and of the American Federation of the Leagues for the Hard of Hearing. He was Secretary of the Board of Trustees of the American Medical Association since 1933 and had been Treasurer from 1922-1933.

During the later years of his life he was especially interested in the hard of hearing, and was active in the American Medical Association Committee for standardizing audiometers and hearing aids. Organized medicine and the hard of hearing have lost an unquestioned friend and indefatigable worker.

IRA FRANK.

PUBLICATIONS OF DOCTOR AUSTIN A. HAYDEN

1. Simplified Surgical Tonsillectomy. Chicago Medical Recorder, 39:151-160, 1917.

2. Vestibular Rotation Reactions and Routine Otolaryngologic Examination of 3,748 Applicants for Aviation Section of Signal Corps of U. S. Army. ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 28:518, 1919.

3. Enucleation of the Eyeball Under Local Anesthesia. Am. J. Ophth., 3:11: 447-449, 1919.

4. Hemophiliac Type Hemorrhage-Treatment by Transfusion. Ann. Otol. and Laryngol., 29:37 (March), 1920.

5. Local Anesthetics: The Prevention and Treatment of Poisoning Therefrom. Tr. Am. Acad. Ophth., 27:76-84, 1922.

6. Auscultation and Percussion in Diagnosis of the Accessory Nasal Sinus Disease. Preliminary report. J. A. M. A., 87:1390-1399 (Oct. 23), 1926.

7. Otolaryngologist in General Hospital Internship. J. A. M. A., 91:443-446 (Aug. 18), 1928; also in Tr. Sect. Laryng., Otol. & Rhinol., A. M. A., pp. 35-45, 1928.

8. Agranulocytosis. Ill. Med. J., 62:537-540 (Dec.), 1932.

9. Presentation of Instruments. Autotympanoscopy. Tr. Am. Laryng., Rhin. & Otol. Soc., 38:307, 1932.

10. Theory of Focal Infection (Concept). Tr. Am. Laryng., Rhin. & Otol. Soc., 38:13-15, 1932.

11. Institution of Medicine. Memphis M. J., 10:6 (Jan.), 1935.

12. Audiometers and Hearing Aids. J. A. M. A., 110:723-725 (Mar. 5), 1938.

13. Hearing Aids from Otologists' Audiogram. J. A. M. A., 111:592-96 (Aug.), 1938; also in Tr. Sect. Laryng., Otol. & Rhinol., A. M. A., pp. 117-130, 1938.

14. Zinc Sulfate Intranasal Spray Technic in Poliomyelitis Prevention. M. Clin. North. Am., 22:107-109, 1938.

15. Hearing Aids from Otologists' Audiogram. Tr. Sect. Laryng., Otol. & Rhinol., A. M. A., pp. 117-130, 1938.

16. Hear and Now. Hygeia., 18:489 (June), 1940.

17. Hearing Aids. Tube or Carbon? J. A. M. A., 115:191-193 (July 20), 1940.

18. When Hearing Aids Should Be Used. Illinois M. J., 78:442-444 (Nov.), 1940.

Books Received

The Author-Publisher-Printer Complex.

By Robert S. Gill. 12mo. 76 pages. The Williams & Wilkins Co., 1940.

If many an editor had his way, many a medical contributor would read this small volume (reading time: well under an hour) and take it to his heart.

A great number of medical authors who should know better are content to dictate their articles to the secretary and send them to the publisher unread, on the principle that grammar, punctuation and style are the other fellow's job, and that editors are paid to look after such things.

This work explains very simply the separate functions of author and publisher. It explains why editors who have every desire to retain the author's flavor and style, are often compelled to sacrifice both because the presentation is second rate as it stands and unworthy of the publication in which it is to appear. It explains further the requirements of fair copy and points out that changes on the galley sheet are costly which might have been made without expense on the manuscript.

It takes up in some detail the handling of illustrations, setting forth, for example, what occurs when the author pastes his legends on the backs of his illustrations, what happens when notes are made with a hard pencil on the backs of unmounted prints, and how reductions are indicated.

Recommended reading for all medical authors.

Index of Authors

ARBUCKLE, M. F., 821.

BERNHEIMER, L. BENNO, 418.
Bowers, W. C., 1036.
Bowman, Robert J., 225.
Bozer, Hermann E., 1091.
Breeding, Earle G., 141.
Brighton, G. R., 1070.
Brownell, D. H., 551, 765, 973.
Brunner, Hans, 688.
Bunch, C. C., 359.
Burke, B. Russell, 924.

CAMPBELL, EDWARD H., 447.
Canfield, Norton, 248.
Childrey, J. H., 709.
Cinelli, Albert A., 912.
Clerf, Louis H., 793.
Cohen, Lee, 410.
Converse, John M., 895.
Cordray, David P., 160.
Cummings, George O., 801.
Cutler, Abraham, 736.

DAMITZ, J. C., 996.
Davison, F. W., 1083.
Dean, L. W., Jr., 76.
Dill, J. Lewis, 996.

FIGI, FREDERICK A., 394.
Ford, W. A., 965.
Frank, Ira, 113.
Fry, Kenneth E., 967.

GOLDEN, M. GERARD, 1032.

HAGENS, E. W., 168.
Hansel, French K., 579.
Heatly, Clyde A., 785
Henry, L. Dell, 519.
Hill, Frederick T., 628.
Hirsch, Caesar, 232.
Hughson, Walter, 262, 368, 384.

JACKSON, C. L., 1048.
Jesberg, Simon, 1045.
Johnson, Charles I., 755.
Johnson, Louis G., 1055.
Jones, Arthur C., 713.

KENNON, W. G., 540.
Kos, Clair M., 151, 510.
Kuhn, Hugh A., 519.

LEV, MAURICE, 113
Lillie, Harold I., 38.
Lindsay, J. R., 661.

McCASKEY, C. H., 199.
McDougall, William L., 744.
McReynolds, George S., Jr., 1048.
Malis, Sol, 239.
Marshall, L. R., 961.
Martin, Hayes E., 728.
Matis, E. I., 559, 991.
Maxwell, J. H., 973.
Mellinger, W. J., 438.
Mosher, Harris P., 3.
Mousel, Lloyd H., 36, 446.
Myerson, Mervin C., 177, 1064.

NEFFSON, A. HARRY, 211.
Novak, Frank J., Jr., 526.

PALMER, R. S., 755.
Parkinson, Sidney N., 1023.
Parris, H. M., 1008.
Pastore, Peter N., 38.
Pearlman, Samuel J., 805.
Pembleton, W. E., 797.

Penta, Arthur Q., 783.
Perrone, Joseph A., 528.
Poe, David L., 771.
Pond, C. W., 500.
Popp, Walter C., 749.
Putney, F. J., 967.

RICHARDS, LYMAN G., 776.
Rindskopf, Wallace, 76.
Roberts, Sam E., 1044.
Rubin, Herman W., 736.
Rusk, Howard A., 76.

SCHALL, LeROY A., 1055.
Segal, H. I., 1008.
Selfridge, Grant, 52, 674.
Seltzer, A. P., 219.
Seydell, Ernest M., 1061.
Shumaker, Phil W., 1038.
Simonton, Kinsey Macleod, 80.
Simpson, W. L., 949.
Slaughter, Earl C., 130.
Smiley, William A., 556.
Smith, Josiah E., 490.
Som, Max L., 211.
Steinberg, Arthur, 1008.
Stinson, William D., 536.

THACKER, E. A., 939.

VADALA, A. J., 291.
Vance, L. Alexander, 755.
Venable, John H., 761.
Vinson, Porter P., 797.
Viole, Pierre, 639.

WALDAPFEL, RICHARD, 647.
Wall, John W., 688.
Walsh, Theodore E., 875.
Warren, Edwin D., 543.
Wells, Walter, 99, 427.
Williams, Henry L., 466, 749.
Williams, R. I., 949.
Witting, E. G., 368.
Wolfe, Michael M., 700.

Index of Titles

ABSCESS, Lung, Bronchoscopic Treatment of. M. F. Arbuckle, 821.

Abscess. Temporo-Sphenoidal Brain Abscess with Operation and Recovery. W. L. McDougall, 744.

Abscess, Tuberculous Periesophageal, Producing Stenosis: Report of a Case. Louis H. Clerf, 793.

Allergy, Nasal, Results of Potassium Therapy in. Howard A. Rusk, L. W. Dean, and Wallace Rindskopf, 76.

Allergy of the Upper and Lower Respiratory Tracts in Children. F. K. Hansel, 579.

Anesthesia, Endo-Tracheal, Polypoid Granuloma of the Larynx Following. Wm. A. Smiley, 556.

Anesthesia for Peroral Endoscopy. C. L. Jackson and G. S. McReynolds, Jr., 1048.

Anesthesia, Local, Methods for Producing, for Tonsillectomy, Intranasal Operations and Operations on the Paranasal Sinuses. Harold I. Lillie, Peter N. Pastore, and Lloyd H. Mousel, 38.

Anomaly, Congenital, of the Aorta, Dyspnea Due to. LeRoy A. Schall and Louis G. Johnson, 1055.

Antrum, Lavage of the, with a Blunt Instrument Through the Middle Meatus. W. C. Bowers, 1036.

Aorta, Dyspnea Due to Congenital Anomaly of the. LeRoy A. Schall and Louis G. Johnson, 1055.

Atelectasis, Postoperative, Bronchoscopy as a Treatment of. J. A. Perrone, 528.

Atresia of Trachea Following Injury: Dilatation with Core Molds. George O. Cummings, 801.

Atresias, Nasal: A Surgical Critique. Albert A. Cinelli, 912.

Auditory Canal, External, Carcinoma of the. R. J. Bowman, 225.

Auditory Canal, External, Report of Two Cases of Pedunculated Osteoma of the. W. G. Kennon, 540.

BIOPSY Forceps, Presentation of. Sam E. Roberts, 1044.

Black Tongue: Report of Two Cases. L. R. Marshall, 961.

Bronchi. Tuberculous Ulcerogranuloma of the Trachea and Bronchi. Mervin C. Myerson, 177.

Bronchial Collapse Without Clinical Signs of External Pressure: Bronchoscopic Observations. H. E. Bozer, 1091.

Bronchitis. Laryngotracheobronchitis. G. R. Brighton, 1070.

Broncho-Pulmonary Tract, Mycotic Infection of the. A. J. Vadala, 291.

Bronchoscopy as a Treatment of Postoperative Atelectasis. Report of Ninety Cases. Joseph A. Perrone, 528.

Bronchoscopy in Tuberculous Children. M. C. Myerson, 1064.

Bronchoscopy, Non-Fogging Spectacles for. Arthur Q. Penta, 783.

Bronchoscopy. Presentation of New Instruments. Simon Jesberg, 1045.

Bronchus. Ulceration of Tuberculous Hilar Lymph Node into Lumen of Bronchus, with Bronchoscopic Removal. Porter P. Vinson and W. E. Pembleton, 797.

CANCER of the Larynx, Selection of Treatment for. H. E. Martin, 728.

Carcino-Sarcoma, So-called, of the Esophagus. S. J. Pearlman, 805.

Carcinoma, Epidermoid, of the Nasopharynx Occurring in Two Young Brothers. Wm. D. Stinson, 536.

Carcinoma of the External Auditory Canal, Middle Ear and Mastoid. R. J. Bowman, 225.

Carcinoma of the Larynx. L. B. Bernheimer, 418.

Carcinoma, Primary, in the Eustachian Tube. Phil W. Shumaker, 1038.

Carcinoma, Primary, of the Eustachian Tube. Durwin H. Brownell, 551.

Carcinoma, Primary, of the Frontal Sinuses. Earle G. Breeding, 141.

Carcinomatosis of the Nasal Mucous Membrane (Fatal Hemorrhage After Puncture of Maxillary Sinus). Hans Brunner and John W. Wall, 688.

Carcinosarcoma of the Larynx. Ira Frank and Maurice Lev, 113.

Cardiospasm, Intractable, The Surgical Treatment of: A Report of Two Cases. Clyde A. Heatly, 785.

Chondroma and Fibroma of the Superior Maxilla: Report of Two Cases. D. H. Brownell, 765.

Cold. Intranasal Vaccine Spray: Its Use in Prophylaxis Against the Common Cold. Theodore E. Walsh, 875.

DEAFNESS, A Summary of Round Window Graft Operations Performed For. Walter Hughson, 384.

Deafness, Chronic Progressive, Results in the Labyrinth Fistulization Operation for. E. H. Campbell, 447.

Deafness, Chronic Progressive, With Special Reference to Estrogenic Substances (A Further Contribution). Grant Selfridge, 52.

Deafness, Pathology of the Inner Ear in a Case of, from Epidemic Cerebrospinal Meningitis. E. W. Hagens, 168.

Deafness, The Type of, Which Responds to Fistulization of the Otic Capsule. Norton Canfield, 248.

Deafness, The Type of, Which Responds to Fistulization of the Otic Capsule. Walter Hughson, 262.

Dorsum, Advantage of Mixed Bone and Cartilage Grafts in Correction of Saddle Nose and Other Depressed Deformities of the. Lee Cohen, 410.

Dyspnea Due to Congenital Anomaly of the Aorta. LeRoy A. Schall and Louis G. Johnson, 1055.

EAR, A Report on the Use of Urea in Suppurative Conditions About the. A. P. Seltzer, 219.

Ear. A Survey of the Relation Between Nutrition and the Ear. Grant Selfridge, 674.

Ear, Middle, Bacteriological Studies of Acute Infections of the L. Dell Henry and Hugh A. Kuhn, 519.

Ear, Middle, Carcinoma of the. R. J. Bowman, 225.

Ear, Sardoidosis of the External. David L. Poe, 771.

Ear. The Use of Human Convalescent Scarlet Fever Serum in Streptococcic Infections Involving the Ear, Nose and Throat. Pierre Viole, 639.

Endoscopy, Peroral, Anesthesia for. C. L. Jackson and G. S. McReynolds, Jr., 1048.

Esophagus, Center of Ossification of the Sternum Mistaken for a Foreign Body in the. E. M. Seydell, 1061.

Esophagus, So-called Carcino-Sarcoma of the. S. J. Pearlman, 805.

Estrogenic Substances, Chronic Progressive Deafness With Special Reference to, A Further Contribution). Grant Selfridge, 52.

Ethmoids, Left Posterior, Air Rifle Buck Shot in: Operative Removal. M. G Golden, 1032.

Eustachian Tube, Carcinoma, Primary, in the. Phil W. Shumaker, 1038.

Eustachian Tube, Primary Carcinoma of the. D. H. Brownell, 551.

FACIAL Defects, Sulfathiazol Used with Cartilage Implants for Repair of. J. H Childrey, 709.

Facial Nerve, Tumor of the, Within the Mastoid Bone. Clair M. Kos, 151.

Fibroma and Chondroma of the Superior Maxilla. D. H. Brownell, 765.

Fibromas of the Nasopharynx: Juvenile and Cellular Types. Max L. Som, 211.

Fistulization of the Otic Capsule, The Type of Deafness Which Responds to. Norton Canfield, 248.

Foreign Body. Air Rifle Buck Shot in Left Posterior Ethmoids. M. G. Golden, 1032.

Foreign Body in the Esophagus, Center of Ossification of the Sternum Mistaken for a. E. M. Seydell, 1061.

Frontal Bone, Microscopic Changes in Osteomyelitis of the. Clair M. Kos, 510.

Frontal Bone, Osteomyelitis of the, with Report of Thirteen Cases. A. C. Jones, 713.

GRANULOMA, Polypoid, of the Larynx, Following Endo-Tracheal Anesthesia. Wm. A. Smiley, 556.

HEAD, Anatomy of the, An Anatomic "Crown" as an Aid for Postgraduate Study of the. Sol Malis, 239.

Hearing. A Summary of Round Window Graft Operations Performed for Deafness. Walter Hughson, 384.

Hearing, Chronic Progressive Deafness with Special Reference to Extrogenic Substances. Grant Selfridge, 52.

Hearing, Estimation of Improvement in, Following Therapy of Deafness. Walter Hughson and E. G. Witting, 368.

Hearing, Pathology of the Inner Ear in a Case of Deafness from Epidemic Cerebrospinal Meningitis. E. W. Hagens, 168.

Hearing, Results in the Labyrinth Fistulization Operation for Chronic Progressive Deafness. E. H. Campbell, 447.

Hearing Tests, Some Practical Considerations with Regard to. W. A. Wells, 427.

Hearing, Usable. C. C. Bunch, 351.

Hemorrhage, Treatment and Control of, Role of Oxalic Acid and Certain Related Dicarboxylic Acids in Arthur Steinberg, H. I. Segal, and H. M. Parris, 1008.

Hoarseness, Chronic: Report of Three Hundred Consecutive Cases. J. C. Damitz and J. L. Dill, 996.

Hoarseness, The Significance of. Walter Wells, 99.

Hyperparathyroidism, Experimental, and Otosclerosis. Earl C. Slaughter, 130.

Hypertrophy, Giant Follicular, of Nasopharynx: Report of a Case. Frank J. Novak, Jr., 526.

INFECTION, Mycotic, of the Broncho-Pulmonary Tract. A. J. Vadala, 291.

Infections of the Middle Ear, Bacteriological Studies of. L. Dell Henry and Hugh A. Kuhn, 519.

LABYRINTH Fistulization Operation for Chronic Progressive Deafness, Results in the: Report of Cases. E. H. Campbell, 447.

Labyrinthine Reaction, A New: "The Waltzing Test". Caesar Hirsch, 232.

Laryngocele Ventricularis. J. R. Lindsay, 661.

Laryngotracheobronchitis. G. R. Brighton, 1070.

Larynx, Carcinoma of the. L. B. Bernheimer, 418.

Larynx, Carcinosarcoma of the. Ira Frank and Maurice Lev, 113.

Larynx, Chronic Stenosis of the, with Special Consideration of Skin Grafting. F. A. Figi, 394.

Larynx, Polypoid Granuloma of the, Following Endo-Tracheal Anesthesia. Wm. A. Smiley, 556.

Larynx. Selection of Treatment for Cancer of the Larynx. H. E. Martin, 728.

Lipoma, Retropharyngeal. F. J. Putney and K. E. Fry, 967.

Lung. Bronchoscopic Treatment of Lung Abscess: Report of Forty-three Cases. M. F. Arbuckle, 821.

MALIGNANCY of the Nasal Accessory Sinuses with a Report of Two Cases of Primary Carcinoma of the Frontal Sinuses. Earle G. Breeding, 141.

Mastoid, Carcinoma of the. R. J. Bowman, 225.

Maxilla, Superior, Chondroma and Fibroma of. D. H. Brownell, 765.

Meatus, Middle, Lavage of the Antrum with a Blunt Instrument Through the. W. C. Bowers, 1036.

Meniere's Symptom Complex: A Review of the Literature. Kinsey M. Simonton, 80.

Meningitis, Epidemic Cerebrospinal, Pathology of the Inner Ear in a Case of Deafness from. E. W. Hagens, 168.

Mortality Records over a Ten-Year Period, A Study of. F. T. Hill, 628.

Mouth, Pemphigus Localized in the. E. I. Matis, 991.

NASOPHARYNX, An Anatomical Study of the, as a Pathway to the Infra-Carotid Portion of the Petrous Pyramid. B. Russell Burke, 924.

Nasopharynx, Epidermoid Carcinoma of the. Wm. D. Stinson, 536.

Nasopharynx, Fibromas of the. Max L. Som, 211.

Nasopharynx, Giant Follicular Hypertrophy of. Frank J. Novak, Jr., 526.

Nose. Advantage of Mixed Bone and Cartilage Grafts in Correction of Saddle Nose and Other Depressed Deformities of the Dorsum. Lee Cohen, 410.

Nose. Carcinomatosis of the Nasal Mucous Membrane (Fatal Hemorrhage After Puncture of Maxillary Sinus). Hans Brunner and John W. Wall, 688.

Nose. Corrective Surgery of the Nasal Tip. John M. Converse, 895.

Nose. Extensive Intranasal Destruction: A Pathological Specimen. John H. Venable, 761.

Nose. Modification of the Steven's Rubber Nasal Balloon Pack. W. A. Ford, 965.

Nose. Nasal Atresias: A Surgical Critique. Albert A. Cinelli, 912.

Nose, Osteoma of the, and Accessory Sinuses. W. L. Simpson and R. I. Williams, 949.

Nose. The Partial Saddle Nose: A Simplified Technique Without the Use of Implant or Transplant. W. M. Wolfe, 700.

Nose. The Surgical Establishment of Nasal Ventilation. S. N. Parkinson, 1023.

Nose. The Use of Human Convalescent Scarlet Fever Serum in Streptococcic Infections Involving the Ear, Nose and Throat. Pierre Viole, 639.

Nutrition and the Ear, A Survey of the Relation Between. Grant Selfridge, 674.

OSSIFICATION, Center of, of the Sternum, Mistaken for a Foreign Body in the Esophagus. E. M. Seydell, 1061.

Osteoma of the Nose and Accessory Sinuses with Report of Cases. W. L. Simpson and R. I. Williams, 949.

Osteoma, Pedunculated, of the External Auditory Canal, Report of Two Cases. W. G. Kennon, 540.

Osteomyelitis of the Frontal Bone, Microscopic Changes in. Clair M. Kos, 510.

Osteomyelitis of the Frontal Bone with Report of Thirteen Cases. A. C. Jones, 713.

Osteomyelitis of the Skull, The Venous Circulation as a Factor in. W. J. Mellinger, 438.

Otic Capsule, Fistulization of the, The Type of Deafness Which Responds to. Norton Canfield, 248.

Otitis Media, Acute Suppurative: A Statistical Study of 1514 Cases, of Which 896 Were Surgical. J. H. Maxwell and D. H. Brownell, 973.

Otosclerosis, Experimental Hyperparathyroidism and. Earl C. Slaughter, 130.

Oxalic Acid, Role of, and Certain Related Dicarboxylic Acids in Treatment and Control of Hemorrhage. Arthur Steinberg, H. I. Segal and H. M. Parris, 1008.

Oxygen Tents, Some Observations on the Control of Temperature and Humidity in. F. W. Davison, 1083.

PAGET'S Disease, Pituitary Tumor and Abscess of the Sphenoid Sinus: Report of a Case. C. W. Pond, 500.

Pemphigus Localized in the Mouth. E. I. Matis, 991.

Petrous Pyramid, An Anatomical Study of the Nasopharynx as a Pathway to the Infra-Carotid Portion of the. B. Russell Burke, 924.

Pharyngomaxillary Approach to the Tip of the Temporal Bone. Harris P. Mosher, 3.

Plummer-Vinson Syndrome, The. David P. Cordray, 160.

Potassium Therapy, Results of, in Nasal Allergy. Howard A. Rusk, L. W. Dean and Wallace Rindkopf, 76.

President's Address, The. Lyman G. Richards, 776.

Prophylaxis. Intranasal Vaccine Spray: Its Use in Prophylaxis Against the Common Cold. Theodore E. Walsh, 875.

Purpura, Thrombopenic, and the Role of X-ray Treatment to the Spleen. Edwin D. Warren, 543.

RESPIRATORY Tracts, Allergy of the Upper and Lower, in Children. F. K. Hansel, 579.

Retropharyngeal Abscess with Reference to Abnormally Large Percentage of Adult Cases. J. E. Smith, 490.

Retropharyngeal Lipoma. F. J. Putney and K. E. Fry, 967.

Rhinitis, The Treatment of Chronic Obstructive, with Submucosal Injections of Sodium Psylliate. E. A. Thacker, 939.

Roentgen Therapy for Acute Sinusitis. H. L. Williams and Walter C. Popp, 749.

SADDLE Nose, Advantage of Mixed Bone and Cartilage Grafts in Correction of, and Other Depressed Deformities of the Dorsum. Lee Cohen, 410.

Scarlet Fever Serum, The use of Human Convalescent, in Streptococcic Infections Involving the Ear, Nose and Throat. Pierre Viole, 639.

Sarcoidosis of the External Ear. David L. Poe, 771.

Sepsis, Postanginal, Two Unusual Cases of. E. I. Matis, 559.

Seventh Nerve Paralysis, The Etiology and Treatment of. C. H. McCaskey, 199.

Sinus. Carcinomatosis of the Nasal Mucous Membrane (Fatal Hemorrhage After Puncture of Maxillary Sinus). Hans Brunner and John W. Wall, 688.

Sinus. Osteoma of the Nose and Accessory Sinuses. W. L. Simpson and R. I. Williams, 949.

Sinus. Paget's Disease, Pituitary Tumor and Abscess of the Sphenoid Sinus. C. W. Pond, 500.

Sinus Thrombosis, Cavernous: A Report of Three Cases with Autopsy Findings. H. W. Rubin and Abraham Cutler, 736.

Sinuses, Malignancy of the Nasal Accessory, with a Report of Two Cases of Primary Carcinoma of the Frontal Sinuses. Earle G. Breeding, 141.

Sinuses, Paranasal, Diagnosis and Treatment of Chronic Disease of the. H. L. Williams and L. H. Mousel, 466.

Sinusitis, Acute, Roentgen Therapy for. H. L. Williams and W. C. Popp, 749.

Skull, The Venous Circulation as a Factor in Osteomyelitis of the. W. J. Mellinger, 438.

Sodium Psylliate, The Treatment of Chronic Obstructive Rhinitis with Submucosal Injections of. E. A. Thacker, 939.

Spectacles, Non-Fogging, for Bronchoscopy. Arthur Q. Penta, 783.

Stenosis, Chronic, of the Larynx with Special Consideration of Skin Grafting. Frederick A. Figi, 394.

Stenosis. Tuberculous Periesophageal Abscess Producing Stenosis. Louis H. Clerf, 793.

Sternum, Center of Ossification of the, Mistaken for a Foreign Body in the Esophagus. E. M. Seydell, 1061.

Streptococcic Infections Involving the Ear, Nose and Throat, The Use of Human Convalescent Scarlet Fever Serum in. Pierre Viole, 639.

Subepithelial (Reinke's Layer of the Vocal Cords, Pathology of the. Richard Waldapfel, 647.

Sulfathiazol Used with Cartilage Implants for Repair of Facial Defects. J. H. Childrey, 709.

TEMPORAL Bone, the Pharyngomaxillary Approach to the Tip of the. Harris P. Mosher, 3.

Throat. The Use of Human Convalescent Scarlet Fever Serum in Streptococcic Infections Involving the Ear, Nose and Throat. Pierre Viole, 639.

Thrombopenic Purpura and the Role of X-ray Treatment to the Spleen. Edwin D. Warren, 543.

Thrombosis, Cavernous Sinus. H. W. Rubin and Abraham Cutler, 736.

Tonsillar Tissue, The Effect of X-Radiation on. C. I. Johnson, R. S. Palmer, and L. A. Vance, 755.

Trachea, Atresia of, Following Injury: Dilatation with Core Molds. George O. Cummings, 801.

Trachea. Laryngotracheobronchitis. G. R. Brighton, 1070.

Trachea. Tuberculous Ulcerogranuloma of the Trachea and Bronchi. Mervin C. Myerson, 177.

Tuberculous Children, Bronchoscopy in. M. C. Myerson, 1064.

Tumor of the Facial Nerve Within the Mastoid Bone. Clair M. Kos, 151.

Tumor, Pituitary, Paget's Disease, and Abscess of the Sphenoid Sinus. C. W. Pond, 500.

Tongue, Black. L. R. Marshall, 961.

ULCERATION of Tuberculous Hilar Lymph Node into Lumen of Bronchus, with Bronchoscopic Removal. Porter P. Vinson and W. E. Pembleton, 797.

Ulcerogranuloma, Tuberculous, of the Trachea and Bronchi. Mervin C. Myerson, 177.

Urea, A Report on the Use of, in Suppurative Conditions About the ear. A. P. Seltzer, 219.

VACCINE. Intranasal Vaccine Spray: Its Use in Prophylaxis Against the Common Cold. Theodore E. Walsh, 875.

Venous Circulation as a Factor in Osteomyelitis of the Skull. W. J. Mellinger, 438.

Vocal Cords, Pathology of the Subepithelial (Reinke's) Layer of the. Richard Waldapfel, 647.

"WALTZING Test, The", A New Labyrinthine Reaction. Caesar Hirsch, 232.

X RADIATION, The Effect of, on Tonsillar Tissue. C. I. Johnson, R. S. Palmer, and L. A. Vance, 755.

X-ray Treatment. Thrombopenic Purpura and the Role of X-ray Treatment to the Spleen. Edwin D. Warren, 543.

V
C
O
P
CT
S
T
A
SH
CA